Modelling aphid populations that are resistant to a fungal pathogen

White, Steven

Award date:
2004

Awarding institution:
University of Bath

Modelling Aphid Populations that are Resistant to a Fungal Pathogen

submitted by

Steven White

for the degree of Doctor of Philosophy

of the

University of Bath

2004

ProQuest LLC
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106-1346

Acknowledgements

I would like to thank the following the following people for their support over the duration of my PhD:

- Dr. Jane White for initially offering me the project and for her continued support and encouragement throughout my time at Bath. Without Jane's guidance I would have found completing my PhD a far more difficult and less enjoyable experience.
- Dr. Keith Charnley for initially offering me the project and for support on all things biological.
- BBSRC for their three years of funding.
- My parents who have encouraged, supported and helped in numerous ways throughout my life.
- Dr. JF Williams who has given me excellent advice and friendship. I would also like to thank JF for the many interesting and hotly debated mathematical discussions.
- Dr. Andy Holt for his friendship and our discussions on theses styles.
- Dr. Nick Britton for his help on mathematical technicalities.
- Dr. Andy White for his help on Adaptive Dynamics.
- My friends, especially Andy Whittle, Jörg Berns-Müller, Fas Yousaf, Damien Harwin and Alex Cox.

Summary

The aim of this thesis is to investigate the dynamical behaviour of two groups of aphid, one that is susceptible to a fungal pathogen, and the other being resistant, using deterministic mathematical models.

Chapter 1 concerns itself with the biological details of aphids, control techniques and in particular fungal pathogens and resistance. This chapter is motivational and is intended to introduce the area of pest insect management, and in particular the problems arising from resistance.

In Chapter 2 I formulate a discrete-time model to investigate the impact of competition between the two strains of insect, and the effect of externally applying the fungal pathogen to the system. The model outcomes are discussed in terms of control of the insect species. The model is then extended to include spatial effects.

Following on from the work carried out in Chapter 2, Chapter 3 considers the implications of using a time-dependent spraying strategy where the control is applied to the system at regular intervals, as opposed to assuming a continuous application of the control. The chapter also compares the fungal pathogen with a chemical insecticide. Analysis is carried out on the long-term behaviour of the models, and pays particular attention to the time between successive applications of control.

Chapter 4 is primarily on adaptive dynamics, and the evolution of resistance. I discuss what effects the externally applied pathogen has on the evolutionary behaviour.

Since many species of aphid can over-winter, a discrete-time model is presented in Chapter 5. The long-term behaviour of the model is studied and results are discussed in terms of the contributions from the susceptible and resistant insects.

In Chapter 6 I present a general framework for the analysis of coupled map lattices. I mainly concern myself with the analysis of dispersal-driven instabilities, and apply my results to the model discussed in Chapter 5.

Contents

Chapter 1

Biological Background

It is well understood that common agricultural pests play a significant role in crop production. Although humans clearly dominate the world in terms of our impact on the environment, the class Insecta can claim to be as important. Insects are humanity's greatest rival for the world's food resources by either eating the plants cultivated for food, or acting as vectors of disease-causing organisms which subsequently damage the crop.
In this introduction, I outline some of the problems caused by insect pests, introduce the aphid as a specific pest, discuss types of pest control, introduce fungal pathogens as a specific type of biocontrol and finally discuss the issue of resistance to the control method.

1.1 The Problem

A pest insect is defined (in the widest sense) as an insect that causes harm to humans, their livestock, crops or possessions (Hill 1997), and is given a pest status when the insect (population) causes a particular level of damage. If an insect species has members that are regularly of pest status then we may refer to the species as a pest species. Example of pest species include the Colorado Beetle and the Tsetse Fly.
Insect pests can be classified into five distinct categories

1. Medical pests
2. Veterinary pests
3. Household and stored products pests
4. Forestry pests
5. Agricultural pests.

For the purpose of this thesis I will only concentrate on agricultural pests, where agriculture refers to the cultivation of plants for food and products such as potatoes, rice, cotton, rubber, and ornamental plants etc.
Direct damage occurs when the insect pest causes visible harm to the plant. One obvious form of direct damage to the plant is caused by the insect feeding. Generally insects feed on leaves, buds, stems, roots, fruits, and seeds, as well as on plant tissue in various stages of decay (Evans 1984). Phytophagous insects feed externally or internally, as borers or leaf miners; they produce galls and other distortions; sucking insects drink plant juices. Some insects are restricted to feeding from one type of plant species (monophagous) while others may have a variety of plants in their diet (polyphagous).
When the insect pest does not cause direct discernible harm to the body of the host plant the damage is called indirect damage. By far the most important form of indirect damage is when the insect acts as a vector for a parasite or pathogen. The simplest form of parasite/pathogen dispersal is known as mechanical transmission, whereby the insect picks up the parasite or pathogen from the host plant whilst feeding, and then deposits it on to a new host or contaminates the food of the host. In agriculture, almost all viral diseases are spread by feeding insects (Hill 1997), such as aphids and leafhoppers.
Another form of dispersal is known as biological transmission. This is when the parasite or pathogen undergoes a developmental stage inside the insect vector. In many cases the insect is more than just a vector and is in fact an intermediate host which plays an important part in the parasite or pathogen life-cycle.
The reason that pest insects are such an important problem is that their reproductive potential is massive and often they can accumulate into large numbers in a short period of time. It has been estimated that 10^{18} insects are alive at any one time in the world (Hill 1997). A large locust swarm may contain one billion insects, in an infested agricultural crop. There can be up to 2×10^6 sycamore aphids (*Drepanosiphum plantanoidis*) on a 20m sycamore tree (Hill 1997). In agriculture, it has been estimated that pest insects and plant pathogens destroy approximately 28% of the potential world food production (Paoletti & Pimentel 2000).

1.2 Aphids

Aphids are insects of the family Aphididae (Aphidae) of the Hemiptera and they are one of the largest and most important groups of pest insects. The family contains approximately 4000 species and can be found worldwide, but are most abundant in north temperate regions (Dixon 1985). Generally aphids are small soft-bodied, plant-sucking insects. Several or all generations comprise parthenogenetic females which do not require fertilisation and are viviparous (i.e. produce live young rather than lay eggs) (Dixon 1985). In some species there are periods of alternating sexual and asexual repro-

Figure 1-1: A close-up of a Peach Aphid *Myzus persicae*

duction and these species are said to show cyclical parthenogenesis, where there may be several generations of parthenogenesis between each bout of sexual reproduction. The aphid life cycle is characterised by a sequence of morphs, called a polyphenism. The morphs differ in behaviour, physiology and structure, and it's due to this characteristic that aphids are robust to environmental change.

Host plants are colonised primarily by alate (winged) aphids that have little control over their flight. When in a relatively still air area around the vegetation, aphids can control their landing and respond to visual and/or olfactory (smell) cues. Once settled, the aphid tests the quality of the plant using their antennae and mouthparts. The probing usually tests the structure and chemistry of the surface, and the outermost internal tissue. This initial investigation of the surface rarely uses the aphids stylets. However, when settled the aphid's stylets probe deep into the plant tissue to get at the phloem (sap). During feeding, aphid saliva is secreted and can be severely toxic to the host (Roditakis 1999).

Phloem sap is rich in sugars but relatively poor in amino acids, which are essential for growth, and therefore aphids must ingest large amounts of sap in order to ingest sufficient amounts of protein (Dixon 1985). They also rely on intracellular symbiotic bacteria (*Buchnera*) to provide essential amino acids, nutrients in short supply in the aphid diet of plant phloem sap. Most of the sap is excreted as droplets of honeydew which can cover the leaves, fruit and vegetables. This rich source of sugar attracts other insects such as ants, and forms an ideal substrate for the growth of moulds (Roditakis 1999).

Most species of aphid, at all stages of development, move about the surface of the host plants and between adjacent plants. This slow dispersal is also coupled with a faster

Figure 1-2: Galls caused by *Pemphigus* sp. aphids

dispersal, which is a characteristic of winged morphs. The development of alate aphids is usually a response to crowding and/or poor host quality.

Direct damage to the host plant caused by aphids usually comes in the form of competition for nutrients and water stress, which can result in reducing the final yield of crop. Moreover, the common response of plant tissue when pierced by the stylet is to form necrotic spots, which can reduce product quality and value (Roditakis 1999). Some aphids induce the development of local structural abnormalities of their host plants (Dixon 1973) (see Figure 1-2). In true galls (as opposed to pseudo-galls, where the galls do not completely close) the plant tissue grows around and completely encloses the aphid and its progeny. Later in the season the gall splits open and the aphids search for a new host. When some aphids feed they cause leaves of the terminal shoots to roll and curl, protecting the aphids inside from natural enemies, weather and pesticides. One explanation as to why this happens is that as the aphids feed on the underside of the leaf, they cause damage to developing cells and clogging and disruption to the vascular system, then as the leaf develops the topside of the leaf develops faster than the bottomside, thus causing the leaf to curl downwards.

The major damage caused by most aphids is due to transmission of viruses. More plant viruses are transmitted by aphids than by any other group of animals (Dixon 1973). Aphids are very efficient virus vectors because of the needle like structure of their maxillary stylets that penetrate deep into the plant tissue (Roditakis 1999). Aphids can carry viruses and pathogens which are retained for life (persistent) or that are lost well before death (non-persistent or semi-persistent) (Harris & Maramorosch 1982).

1.3 Control Methods

There are many different methods of pest control available, depending on the type of pest. In this section I discuss some methods for general types of pest.

Physical Methods These types of method are often desirable as they do not rely on synthetic or toxic agents. Some methods, such as hand picking caterpillars on young fruit trees, can be effective but incur high labour costs. Other types of physical methods include providing netting/shelters and using extreme temperatures.

Cultural Control Generally these types of method do not give high levels of pest control but are usually inexpensive. Typical forms of this method are optimal growing conditions, specific times of sowing and harvesting, crop rotations and intercropping.

Breeding/Genetic Methods Host resistance to pest attack is shown by all groups of plants. The idea is to selectively breed or genetically modify (GM) the host plant in order to enhance the resistance to the pest (and also improve the appearance, size, speed of growth and taste of the crop etc). However, there has been much concern in using GM products (see Pollan (1998), Anon (2002) and Cruywagen, Kareiva, Lewis & Murray (1996) for examples) with the possible risks to human health and to the environment.

Biological Control Biological control (biocontrol) is usually defined as the deliberate supplementation of existing natural control by the introduction of selected predators, parasites or pathogens to the pest/crop ecosystem. Important groups of natural control include birds, spiders, pathogens and many types of insects including wasps and ants. It does appear that in many cases common crop pests are subject to an 80-90% loss of population by the local natural enemies (Hill 1997), and so it is important that this predation is preserved when adopting a control programme. A specific type of biocontrol, namely the use of fungal pathogens, is discussed in Section 1.4. Another common scheme is insect sterilisation, whereby males are sterilised (without affecting their sexual behaviour) and released so that they outnumber normal males. One other type of natural control uses semiochemicals and in particular, pheromones. These may be used to attract and trap the insects or to disrupt sexual behaviour.

Chemical Control Chemical poisons that kill insects are known as insecticides, while acaricides kill mites and ticks. The main modes of action for insecticides are:

Repellents Designed to keep pest insects away from the host.

Antifeedants Chemicals which block part of the feeding response.

Fumigants Volatile chemicals that vaporise, with the resultant toxic gas killing the pest.

Smokes Insecticidal powder mixed with combustible material and dispersed as smoke.

Stomach Poisons Poisons that are sprayed on foliage or mixed with bait to encourage ingestion. These were the earliest forms of insecticide.

Contact Poisons Poisons that are sprayed onto the crop and are typically absorbed directly through the cuticle of the pest. These types of poisons can be long or short lived.

Systemic Poisons Poisons are taken up by the plant and kill the pest when it feeds. They are mostly used against sap-sucking insects such as aphids.

It has been estimated that without insecticides, crop losses due to pests might increase by 30%, and it has also been estimated that insecticides return about $4 per dollar invested in insecticide applications (Paoletti & Pimentel 2000). Despite the undoubted success of chemical insecticides there has been much concern over their use, particularly their effect on the environment. Problems include safety for humans and other non-target organisms, decreased activity of natural enemies, decreased biodiversity in managed ecosystems and pesticide residues in food (see the Soil Association (`www.soilassociation.org`)). There is also an extensive problem with pest insects becoming resistant to insecticides, which is discussed in Section 1.5.

As Hill (1997) discussed, when considering which method of control to use, the main factors to be considered can be grouped under the following headings: expected damage to the host, degree of risk to the host, nature of the pest complex, economic factors, and biological/ecological factors.

1.4 Fungal Pathogens

The development of insecticide resistance and concerns over environmental impact, increased attention on alternative forms of insect pest control (Charnley 1997).
Like other natural enemies, insect pathogens can exert considerable control on target pest populations. Various natural enemies have been tested as candidate biocontrol agents. However, very few have been widely accepted.
Aphid reproductive capacity is very high. Some aphids produce hundreds of offspring (Dixon 1985) and only few natural enemies, except for micro-organisms, have similar reproductive potentials. Successful biological control must therefore be based on repeated releases (Roditakis 1999).

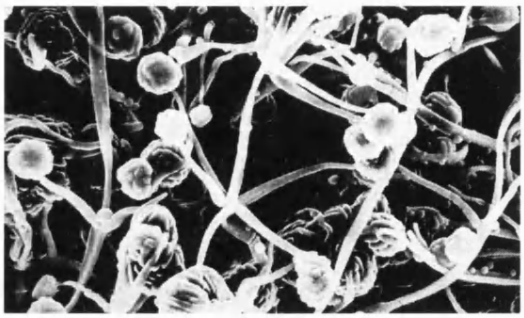


Figure 1-3: Spores (conidia) of the entomopathogenic fungus *Verticillium lecanii*

In most species of entomopathogenic fungi, access to the host is through the cuticle and may involve complex biochemical interactions between the host and the fungus before germination, penetration, growth, and reproduction of the fungus can occur (Lacey, Frutos, Kaya & Vail 2001). Therefore insect pathogenic fungi do not have to be ingested, unlike most entomopathogens (Charnley 1997). This has the advantage that they can infect non-feeding stages such as eggs and pupae.

Fungal invasion of the insect host starts with the spores of the fungus binding to the insect cuticle on which it germinates. See Figure 1-3 for an example of fungal spores. After a period of horizontal growth on the surface of the cuticle, the fungus initiates cuticle penetration. Once the fungus breaks through the cuticle and underlying epidermis, it may grow profusely in the blood, in which case death of the insect is probably caused by starvation or physiological/biochemical disruption. Alternatively, insecticidal secondary metabolites may contribute to the demise of the insect and, in this case, extensive growth of the fungus may occur on the cadaver of the host. For many fungi the reality is probably somewhere between these two extremes (Charnley 1997). The life cycle is then completed when the fungus sporulates on the cadaver of the host insect. Under certain conditions, the fungus may produce aerial spores which may allow horizontal or vertical transmission of the disease within the host insect population. Some spores may be able to survive for long periods in adverse conditions in the dead insect. Environmental conditions, particularly humidity and temperature and to a lesser extent light and air movement, are very important in the infection and sporulation of entomopathogenic fungi (Tanada & Kaya 1993). This has lead to major constraints on the use of fungi for insect control. However, there is massive potential for the use of fungal pathogens in insect pest control.

Verticillium lecanii is one of the most important and common pathogens of scale insects and aphids in tropical and semitropical environments (Roditakis 1999). It has been used in crop protection in glasshouses with good results. Under the name Vertalec^{TM}, *V. lecanii* has been developed as the only commercially produced myco-insecticide against aphids.

1.5 Resistance

Resistance is defined as the development of an ability in a strain of insects to tolerate doses of toxicants or pathogens, which would prove lethal to the majority of individuals in a normal (susceptible) population of the same species.
Resistance to pesticides in arthropod pests is a significant economic, ecological and public health problem. More than 500 arthropod species have become resistant to insecticides and acaricides, with many species having become resistant to the major classes of such products (Hoy 1998). Some strains have become so resistant to a given insecticide that they can survive exposure to virtually any dose (Scott 1990).
In glasshouses, some aphids such as *Myzus persicae* reproduce continuously by parthenogenesis under a strong selection pressure from insecticides. The result is a resistant aphid and the development of the resistance mechanism is much faster than compared to field populations. Moreover, the resulting resistance is also considerably stronger than that of the resistant field aphid (Roditakis 1999).
It was generally thought that resistance to natural biocontrols would not occur, and initially this did seem to be the case; however, it is now clear that some resistance to these products is developing (Hill 1997). For example, resistance has been found in natural populations of pea aphids (*Acyrthosiphon pisum*) to the pathogenic fungus *Erynia neoaphidis* (Milner 1985, Milner 1982, Ferrari & Godfray 2003). There has also been evidence that it is possible to induce resistance, for example in Milks & Theilmann (2000) the authors were able to select for resistance to a baculovirus in cabbage loopers. Although induced resistance to biocontrols in insect pests has only been shown in laboratory conditions to date, the question as to whether resistance to biocontrols occurs in the fields and glasshouses remains unanswered.

Chapter 2

Continuous-Time Model

2.1 Motivation and Background

2.1.1 Chapter Outline

This chapter is split into two sections. The first section presents a simple continuous-time model to investigate the competition between a susceptible and resistant strain of aphid under the attack of a fungal pathogen. I consider the spatially homogeneous case which results in a model consisting of system of coupled nonlinear ordinary differential equations (ODE's). The long-term behaviour of this model is then analysed in detail. The model formulated in the first section is then spatially extended using a lattice ODE approach. Simulations are then carried out.

2.1.2 Motivation

In warmer more constant climates aphids may not produce sexual morphs. In fact it is well known that of the factors in the environment of an aphid, day-length, temperature and the quality of the host are important in the induction of sexual morphs (Dixon 1973). Therefore, in environments such as greenhouses where the conditions remain nearly constant, aphids are likely to remain as parthenogenetic morphs, which reproduce continuously at the population level. Moreover, in temperate climates, where the summer may be long, the aphids may reproduce parthenogenetically for many months before sexual morphs are formed in the autumn (Dixon 1973).
This almost constant reproduction can be well approximated by a continuous time model (as opposed to a discrete time model).

2.1.3 Background Reading

Continuous Time Models

In the past, models have been developed to study host-pathogen (or parasite) systems which include a dynamical description of the free-living infective stages of the pathogen. In Anderson & May (1981) the authors consider in great detail the population dynamics of microparasites (which include viruses, bacteria, protozoans and fungi) and their insect hosts. In particular, in their Model G, the authors include the free-living stage of the microparasite. This model takes the form

$$\begin{aligned}
\frac{dX}{dt} &= a(X+Y) - bX - \nu WX + \gamma Y, \\
\frac{dY}{dt} &= \nu WX - (\alpha + b + \gamma)Y, \\
\frac{dH}{dt} &= rH - \alpha Y, \\
\frac{dW}{dt} &= \lambda Y - (\mu + \nu H)W,
\end{aligned}$$

where $X(t), Y(t), H(t)$ and $W(t)$ are the densities of susceptible hosts, infected hosts, total hosts ($H = X + Y$) and of free-living infective stages of the parasite, at time t respectively and $r = a - b$. This model assumes the mass-action law for disease transmission, that is, the per capita rate of infection for susceptibles is directly proportional to the density of free-living parasites, with transmission coefficient ν.
Other important assumptions in Anderson and May's Model G include:

- No vertical transmission. That is, there is no parasite transmission between parent and unborn offspring.
- Infected individuals can recover from infection at rate γ. As the authors pointed out, for most microparasitic infections of invertebrates there is no recovery. Therefore in the models presented in this thesis, I assume this term to be zero.
- The free-living stages of parasite are lost upon being picked up by the hosts (νWH) or by mortality (μW). When considering a control strategy (i.e. applying large amounts of parasites into the system), the amount of parasites lost to pick-up will be relatively small when compared to the amount released. Therefore, in my models I shall neglect this term.
- In the absence of parasitism, the hosts grow exponentially. Therefore any host population regulation (if it exists) comes from parasitism. Clearly this assumption is unrealistic for many pest species, as competition for resources must play an important role in the dynamics.

Alternative forms of transmission terms (or incidence rates) have been considered in Liu, Hethcote & Levin (1987) and Hochberg (1991) where instead of the standard bilinear form, a nonlinear form $\nu W^p X^q$ is considered. They have shown that models with the nonlinear incidence rates have a much wider range of dynamical behaviours than those with bilinear incidence rates. In the models presented in this thesis, only bilinear forms are considered. This enables the analysis of the models to be less cumbersome, and to obtain tractable ecological results.

As Anderson & May's (1981) paper identifies, it is possible to regulate the host population by the use of a parasite, where the only form of regulation comes from the parasite, and not the host itself. In Holt & Pickering (1985) the authors consider a model with two species that have no self-regulation but are regulated by a common infectious disease. Although similarities are drawn to Lotka-Volterra competition models in the way that the hosts are regulated, if one of these species is resistant to the disease then the regulation is lost.

As discussed in Section 1.5, resistance to a pathogen can be induced by its overuse. If this resistant strain of insect hosts invades the resident susceptible population such that a polymorphism exists, the host population dynamics will significantly change. In both Antonovics & Thrall (1994) and Bowers, Boots & Begon (1994) the authors consider the dynamics of a resistant and a susceptible strain of a self-regulated host species, in the presence of a directly transmitted pathogen (i.e. the pathogen does not have free-living infective stages explicitly modelled). In both studies, the authors conclude that a polymorphism is most likely when the differences between strains are large. These differences are encapsulated by varying degrees of susceptibility and reproduction potentials. Bowers et al. (1994) found that with highly pathogenic pathogens, a susceptible strain may exclude a resistant strain because its higher growth rate is more effective against the pathogen than reduced transmissibility. In both papers, the trade-off with a reduced susceptibility to the pathogen is a reduced intrinsic growth rate and a higher cost to over-crowding. However, as Ferrari & Godfray (2003) indicates, the trade-off may not as easily be attributed. For example, interspecific and intraspecific competitive abilities may vary between the two strains of host.

In this chapter I build upon the work cited above to include

- competition between susceptible and resistant aphids,
- an explicitly modelled free-living fungal pathogen,
- a constant spraying strategy.

Metapopulations, Cellular Automata and ODE Lattices

A metapopulation can be defined as an ensemble of local populations (or subpopulations) which interact through the interchange of individuals through migration

(Hanski & Simberloff 1997). This concept was originally formalised in ecology by Levins (1969, 1970) who developed a simple patch-occupancy model. In this model it is assumed that there are a large number of identical habitat patches, which are either occupied by a certain species or are empty. The model derived is

$$\frac{dp}{dt} = mp(1-p) - ep$$

where $p(t)$ represents the proportion of occupied patches at time t, and m is the migration rate and e is the extinction rate. The model predicts that the species persists if, and only if, the migration rate is greater than the extinction rate. The main criticisms of this model is that the spatial structure and effects are ignored, and that the demographics and density of the species are not taken into account, which may be important to the behaviour of the system as a whole.

There are many types of metapopulations including, cellular automata, coupled map lattices (see Chapter 6) and ODE lattices.

A cellular automaton (CA) usually consists of a regular spatial lattice of cells, each of which can be in any one of a finite number of states. The states of all cells in the lattice are updated simultaneously and the state of the entire lattice advances in discrete time steps. The state of each cell in the lattice is updated according to local rules which may depend on the state of the cell and the neighbouring cells at the previous time step. Ermentrout & Edelstein-Keshet (1993) provides an excellent review of examples of cellular automata that have been used to model biological systems. One downside to the CA is that the number of states for the cell is finite, and so for insect populations for example, these types of models may not be suitable where the dynamics may require variable states.

The idea of ODE lattices is that an ODE system is solved on a lattice domain with associated movement rules. The main problems with ODE lattices are that simulations are often difficult and time consuming to perform, and that there are few analytical techniques available (but see Chow, Mallet-Paret & Shen (1998) and Jansen & Lloyd (2000)). However, they do have some advantages over the CA, since ODE lattices allow for continuous states at each cell, rather than a finite number of states, and since the within cells growth rules are determined by a system of ODEs, the simulations may be more realistic than their CA counterparts.

2.2 The Continuous-Time Model

I start this section by reviewing the classical Lotka-Volterra Competition Model (Murray 1989) for two interacting organisms. I then extend this model to incorporate the free-living fungal pathogen.

2.2.1 Lotka-Volterra Competition Model

In the Lotka-Volterra competition model it is assumed that there are two species (or organisms) dwelling in the same habitat which share (compete for) resources. In my context, these two species are the susceptible and resistant aphids. In the absence of one of the competitors the species grows according to the logistic law. Thus there are two types of competition acting on each of the species, both intraspecific (within species) and interspecific (between species) competition. The model has the form

$$\frac{dS}{dt} = r_S S\left(1 - \frac{S + \alpha_1 R}{K_S}\right) \tag{2.1a}$$

$$\frac{dR}{dt} = r_R R\left(1 - \frac{R + \alpha_2 S}{K_R}\right) \tag{2.1b}$$

where $S(t)$ and $R(t)$ are the densities of the susceptible and resistant aphids respectively at time t, r_S and r_R are the intrinsic growth rates, K_S and K_R are the carrying capacities, and α_1 and α_2 are the competition coefficients (a measure of how much competition a strain exerts on the competing strain).
The Lotka-Volterra model (2.1) has four steady states:

1. The trivial steady state $(S, R) = (0, 0)$.
2. The resistants-eliminated steady state $(S, R) = (K_S, 0)$.
3. The susceptibles-eliminated steady state $(S, R) = (0, K_R)$.
4. The coexistence (non-trivial) steady state $(S, R) = \left(\frac{K_S - \alpha_1 K_R}{1 - \alpha_1 \alpha_2}, \frac{K_R - \alpha_2 K_S}{1 - \alpha_1 \alpha_2}\right)$.

From the linear stability analysis using the Jacobian, it can be shown that:

1. The trivial steady state is never stable. In other words, both strains of aphid will never go extinct simultaneously, since it is assumed that $r_S, r_R > 0$.
2. The resistants-eliminated steady state is stable if, and only if, $K_R - \alpha_2 K_S < 0$. In this case the susceptibles are strong interspecific competitors, and have out-competed the resistants.
3. The susceptibles-eliminated steady state is stable if, and only if, $K_S - \alpha_1 K_R < 0$. In this case the resistants are strong interspecific competitors, and have out-competed the susceptibles.
4. The coexistence steady state is stable if, and only if, $K_R - \alpha_2 K_S > 0$ and $K_S - \alpha_1 K_R > 0$ (which imply that $\alpha_1 \alpha_2 < 1$). In this case both the susceptibles and resistants are weak competitors, and so neither strain is out-competed.

Note that it is possible that both strains of aphid are strong competitors ($K_R - \alpha_2 K_S < 0$ and $K_S - \alpha_1 K_R < 0$), in which case the long-term outcome will depend on the initial densities as to which strain out-competes the other strain.

2.2.2 Model Assumptions and Formulation

I now build upon the Lotka-Volterra model by assuming that:

- In the absence of the fungus, the susceptibles and resistants grow according to the Lotka-Volterra competition model.
- The susceptibles become infected by the fungi at rate β proportional to the number of susceptibles and the amount of free-living fungi in the environment (the mass-action law).
- The resistants are totally resistant to the fungus and so do not become infected.
- Once a susceptible becomes infected, it cannot reproduce nor compete for resources.
- The infecteds do not compete for resources.
- The infected have an induced mortality rate δ.
- On average, the deceased infected release ω free-living fungal spores into the environment.
- The free-living spores have a natural mortality rate d.
- The fungus is externally applied to the system at constant rate a.

These assumptions lead to the model:

$$\frac{dS}{dt} = r_S S\left(1 - \frac{S + \alpha_1 R}{K_S}\right) - \beta SF \tag{2.2a}$$

$$\frac{dR}{dt} = r_R R\left(1 - \frac{R + \alpha_2 S}{K_R}\right) \tag{2.2b}$$

$$\frac{dI}{dt} = \beta SF - \delta I \tag{2.2c}$$

$$\frac{dF}{dt} = \omega\delta I - dF + a \tag{2.2d}$$

where $S(t), R(t), I(t)$ and $F(t)$ are the densities of susceptibles, resistants, infecteds and free-living fungal spores at time t.

2.2.3 Long-term Behaviour

In now consider the long-term behaviour of Model 2.2 in two stages. Firstly I consider the case when $a = 0$, that is, when there is no external application of fungus. This can be thought of as a natural epidemic or as an initial single application of fungus which uses the secondary infection of the fungal pathogen to control the pest aphids (if possible). Secondly I consider the case when $a > 0$, that is, when there is an external application of fungus. I then compare the two cases.

No External Application of Fungus: a = 0

Putting $a = 0$ into Model 2.2 and setting

$$\frac{dS}{dt} = \frac{dR}{dt} = \frac{dI}{dt} = \frac{dF}{dt} = 0$$

it can be shown that six steady states are possible:

1. The trivial steady state

$$(S, R, I, F) = (0, 0, 0, 0).$$

2. The susceptibles-only steady state

$$(S, R, I, F) = (K_S, 0, 0, 0).$$

3. The resistants-only steady state

$$(S, R, I, F) = (0, K_R, 0, 0).$$

4. The susceptibles- and resistants-only steady state

$$(S, R, I, F) = \left(\frac{K_S - \alpha_1 K_R}{1 - \alpha_1\alpha_2}, \frac{K_R - \alpha_2 K_S}{1 - \alpha_1\alpha_2}, 0, 0\right),$$

which is ecologically realistic provided $K_S - \alpha_1 K_R > 0$ and $K_R - \alpha_2 K_S > 0$, or $K_S - \alpha_1 K_R < 0$ and $K_R - \alpha_2 K_S < 0$.

5. The resistants-eliminated steady state

$$(S, R, I, F) = \left(\frac{d}{\beta\omega}, 0, \frac{dr_S}{\omega\delta\beta K_S}\left(K_S - \frac{d}{\beta\omega}\right), \frac{r_S}{\beta K_S}\left(K_S - \frac{d}{\beta\omega}\right)\right),$$

which is ecologically realistic provided $K_S > \frac{d}{\beta\omega}$.

6. The coexistence (non-trivial) steady state

$$(S, R, I, F) = (S^*, R^*, I^*, F^*),$$

where

$$\begin{aligned} S^* &= \frac{d}{\beta\omega}, \\ R^* &= K_R - \alpha_2 \frac{d}{\beta\omega}, \\ I^* &= \frac{dr_S}{\omega\delta\beta K_S}\left(\left(K_S - \frac{d}{\beta\omega}\right) - \alpha_1\left(K_R - \alpha_2\frac{d}{\beta\omega}\right)\right), \\ F^* &= \frac{r_S}{\beta K_S}\left(\left(K_S - \frac{d}{\beta\omega}\right) - \alpha_1\left(K_R - \alpha_2\frac{d}{\beta\omega}\right)\right). \end{aligned}$$

Thus the non-trivial steady state is ecologically realistic provided $K_R - \alpha_2\frac{d}{\beta\omega} > 0$ and $\left(K_S - \frac{d}{\beta\omega}\right) - \alpha_1\left(K_R - \alpha_2\frac{d}{\beta\omega}\right) > 0$. Hence a necessary condition is that $K_S - \frac{d}{\beta\omega} > 0$.

One can now determine the linear stability of these steady states by determining the Jacobian, J, and evaluating it at the respective steady states, where J is given by

$$J(S, R, I, F) = \begin{pmatrix} r_S\left(1 - \frac{2S+\alpha_1 R}{K_S}\right) - \beta F & -\frac{\alpha_1 r_S}{K_S}S & 0 & -\beta S \\ -\frac{\alpha_2 r_R}{K_R}R & r_R\left(1 - \frac{2R+\alpha_2 S}{K_R}\right) & 0 & 0 \\ \beta F & 0 & -\delta & \beta S \\ 0 & 0 & \omega\delta & -d \end{pmatrix}. \tag{2.3}$$

1. For the trivial steady state the Jacobian is

$$J = \begin{pmatrix} r_S & 0 & 0 & 0 \\ 0 & r_R & 0 & 0 \\ 0 & 0 & -\delta & 0 \\ 0 & 0 & \omega\delta & -d \end{pmatrix}.$$

Thus the spectrum of J is $\sigma(J) = \{r_S, r_R, -\delta, -d\}$ and so the steady state is not stable for any parameter combination, since there exist eigenvalues with positive real part. Therefore, it is impossible to completely eradicate the aphids using a single application of fungus.

2. For the susceptibles-only steady state the Jacobian has characteristic polynomial

$$\left(r_R\left(1 - \frac{\alpha_2 K_S}{K_R}\right) - \lambda\right)(-r_S - \lambda)\left(\lambda^2 + (\delta + d)\lambda + \delta(d - \beta\omega K_S)\right) = 0.$$

Therefore it follows that all eigenvalues have negative real part if, and only if,

$$K_R - \alpha_2 K_S < 0$$

and the roots of

$$\lambda^2 + (\delta + d)\lambda + \delta(d - \beta\omega K_S) = 0 \tag{2.4}$$

have negative real part. From the Routh-Hurwitz criteria for quadratics (see Appendix A.2) it follows that (2.4) has eigenvalues with negative real parts if, and only if,

$$K_S < \frac{d}{\beta\omega}.$$

Hence the susceptibles only steady state is stable if, and only if,

$$K_R - \alpha_2 K_S < 0 \quad \text{and} \quad K_S < \frac{d}{\beta\omega}$$

Thus the susceptibles must be stronger competitors than the resistants (which is the same for the Lotka-Volterra model) and in addition the susceptibles steady state must be below some critical threshold in order to stop the fungal pathogen from spreading.

3. For the resistants-only steady state the steady state is stable if, and only if, $K_S - \alpha_1 K_R < 0$. This makes intuitive sense when comparing this stability condition with the stability conditions on the susceptibles only steady state. In order for the resistants to out-compete the susceptibles the resistants just have to be a stronger competitor. However, if the susceptibles want to out-compete the resistants then they must be a stronger competitor and survive the attack from the pathogen.

4. When considering the stability of the susceptibles- and resistants-only steady state, the characteristic polynomial satisfies

$$\begin{aligned} 0 &= \left(\lambda^2 + (\delta + d)\,\lambda + \delta\left(d - \omega\beta\frac{K_S - \alpha_1 K_R}{1 - \alpha_1\alpha_2}\right)\right) \text{ and} \\ 0 &= \left(\lambda^2 + \left(\frac{r_R}{K_R} + \frac{r_S}{K_S}\right)\lambda + (1 - \alpha_1\alpha_2)\,\frac{r_S r_R}{K_S K_R}\,\frac{K_S - \alpha_1 K_R}{1 - \alpha_1\alpha_2}\,\frac{K_R - \alpha_2 K_S}{1 - \alpha_1\alpha_2}\right) \end{aligned}$$

Thus the steady state is biologically realistic (i.e. the steady state is positive) and stable if, and only if,

$$K_S - \alpha_1 K_R > 0, \quad K_R - \alpha_2 K_S > 0, \text{ and } \quad \frac{K_S - \alpha_1 K_R}{1 - \alpha_1\alpha_2} < \frac{d}{\beta\omega}.$$

The first two conditions are the same as those for the Lotka-Volterra model, and the third condition simply says that the susceptible population must be sufficiently low to stop the fungal pathogen spreading.

5. The resistants-eliminated steady state is stable and biologically realistic if, and only if,

$$K_S > \frac{d}{\beta\omega}, \quad K_R - \alpha_2\frac{d}{\beta\omega} < 0, \text{ and } \quad \frac{r_S d}{K_S\beta\omega} + \delta + 2d - \beta\omega K_S > 0.$$

The first condition simply ensures that there are sufficient susceptibles in the population for the pathogen to sustain itself. The second condition says that the susceptibles are strong competitors even in the presence of the pathogen. I have been unable to attach any biological meaning to the third condition.

6. The conditions for the coexistence steady state to be stable can be explicitly found but have no clear biological meaning, and so are not presented here. However, for the coexistence steady state to be biologically realistic, the following conditions must hold:

$$K_R - \alpha_2\frac{d}{\beta\omega} > 0 \quad \text{and} \quad (K_S - \alpha_1 K_R) - \frac{d}{\beta\omega}(1 - \alpha_1\alpha_2) > 0.$$

The first condition states that the resistants must be a strong competitor when the pathogen interacts with the susceptibles. If the two conditions hold, then it is easy to show that they imply

$$K_S > \frac{d}{\beta\omega},$$

in other words there are sufficient susceptibles in the population for the pathogen to exist. Moreover, if the second condition holds and we assume that in the absence of the pathogen, the susceptibles and resistants can coexist (so that $1 - \alpha_1\alpha_2 > 0$) then

$$\frac{d}{\beta\omega} < \frac{K_S - \alpha_1 K_R}{1 - \alpha_1\alpha_2}.$$

Therefore, the density of susceptibles has been reduced with the introduction of the fungal pathogen. Moreover,

$$\frac{K_S - \alpha_1 K_R}{1 - \alpha_1\alpha_2} + \frac{K_R - \alpha_2 K_S}{1 - \alpha_1\alpha_2} > \frac{d}{\beta\omega} + \left(K_R - \alpha_2\frac{d}{\beta\omega}\right) \quad \text{if, and only if,} \quad \alpha_2 < 1.$$

In other words, the total number of aphids when there is no pathogen present is greater than the total when there is pathogen present if, and only if the suscep-

tibles do not exert a relatively large amount of competition on the resistants.

Notice that since

$$K_R - \alpha_2 \frac{d}{\beta\omega} > 0,$$

then

$$K_S - \frac{d}{\beta\omega} > (K_S - \alpha_1 K_R) - \frac{d}{\beta\omega}(1 - \alpha_1\alpha_2).$$

Therefore, when the pathogen can persist, the addition of the resistants has lowered the amount of fungal pathogen in the environment and the density of infecteds.

Since I have not been able to cover the whole of the possible parameter space for this model, there is the possibility of cycles for certain parameter regions.

External Application of Fungus: $a > 0$

Now setting

$$\frac{dS}{dt} = \frac{dR}{dt} = \frac{dI}{dt} = \frac{dF}{dt} = 0$$

in to Model 2.2 it can be shown that there exist four feasible equilibria:

1. The pathogen only steady state

$$(S, R, I, F) = (0, 0, 0, a/d).$$

2. The resistants and pathogen steady state

$$(S, R, I, F) = (0, K_R, 0, a/d).$$

3. The resistants-eliminated steady state

$$(S, R, I, F) = (\hat{S}, 0, \hat{I}, \hat{F}),$$

where $\hat{S}$ satisfies the quadratic

$$A\hat{S}^2 - B\hat{S} + C = 0 \tag{2.5}$$

with

$$\begin{aligned} A &= \omega\beta r_S > 0 \\ B &= dr_S + \omega r_S \beta K_S > 0 \\ C &= dr_S K_S - a\beta K_S \end{aligned}$$

and

$$\begin{aligned} \hat{I} &= \frac{r_S}{\delta}\left(1 - \frac{\hat{S}}{K_S}\right)\hat{S} \\ \hat{F} &= \frac{r_S}{\beta}\left(1 - \frac{\hat{S}}{K_S}\right). \end{aligned}$$

Note that $\hat{S} < K_S$ so that $\hat{I}, \hat{F} > 0$. Now there are two possible solutions to (2.5),

$$\hat{S}_+ = \frac{B + \sqrt{B^2 - 4AC}}{2A} \quad \text{and} \quad \hat{S}_- = \frac{B - \sqrt{B^2 - 4AC}}{2A}.$$

Now $\hat{S}_+ > K_S$ if, and only if,

$$\sqrt{B^2 - 4AC} > 2K_S A - B = r_S\left(\omega\beta K_S - d\right).$$

Thus, if $\omega\beta K_S - d < 0$ then $\hat{S}_+ > K_S$. Conversely, if $\omega\beta K_S - d > 0$ then $\hat{S}_+ > K_S$ if, and only if,

$$B^2 - 4AC > \left(2K_S A - B\right)^2.$$

Then by simplifying the above expression, it is easily seen that $\hat{S}_+ > K_S$. A simular argument shows that $\hat{S}_- < K_S$. Hence $\hat{S} = \hat{S}_-$ is the only feasible solution. Thus the steady state is biologically realistic (i.e. $0 < \hat{S} < K_S$) if, and only if, $C > 0$, or equivalently

$$a < \frac{dr_S}{\beta}. \tag{2.6}$$

A simple calculation shows that the steady state, $\hat{S}$ is a decreasing function of a.

4. The coexistence (non-trivial) steady state is

$$(S, R, I, F) = (S^*, R^*, I^*, F^*)$$

where S^* is given by the quadratic

$$(S^*)^2 - B^* S^* + C^* = 0,$$

and

$$\begin{aligned} B^* &= \frac{d}{\omega\beta} + \frac{K_S - \alpha_1 K_R}{1 - \alpha_1\alpha_2} \\ C^* &= \frac{d}{\omega\beta}\frac{K_S - \alpha_1 K_R}{1 - \alpha_1\alpha_2} - \frac{aK_S}{\omega r_S(1 - \alpha_1\alpha_2)} \\ R^* &= K_R - \alpha_2 S^* \\ F^* &= \frac{r_S}{\beta K_S}\left((K_S - \alpha_1 K_R) - (1 - \alpha_1\alpha_2)\, S^*\right) \\ &= \frac{r_S}{\beta K_S \alpha_2}\left((1 - \alpha_1\alpha_2)\, R^* - (K_R - \alpha_2 K_S)\right) \\ I^* &= \frac{dF^* - a}{\omega\delta}. \end{aligned}$$

Thus if $I^* > 0$ then

$$F^* > \frac{a}{d}. \tag{2.7}$$

Now, if

$$K_S - \alpha_1 K_R > 0 \quad \text{and} \quad K_R - \alpha_2 K_S > 0 \tag{2.8}$$

the non-trivial steady state in the Lotka-Volterra model is positive and stable. Thus in order that the non-trivial steady state is biologically realistic, we get the necessary conditions

$$S^* < \frac{K_S - \alpha_1 K_R}{1 - \alpha_1\alpha_2} \quad \text{and} \quad R^* > \frac{K_R - \alpha_2 K_S}{1 - \alpha_1\alpha_2}. \tag{2.9}$$

If the inequalities in (2.8) were to be reversed, then the inequalities in (2.9) would be reversed.

Under assumption (2.8) there is only one biologically realistic solution to the quadratic for S^* given by

$$S^* = \frac{B^* - \sqrt{(B^*)^2 - 4C^*}}{2}.$$

On the other hand, if the inequalities in (2.8) are reversed then again there is only one biologically realistic solution to the quadratic for S^* but this time it is given by

$$S^* = \frac{B^* + \sqrt{(B^*)^2 - 4C^*}}{2}.$$

One can now determine the stability of these steady states by the Jacobian. Since the case for externally applied pathogen only differs by a constant from the no externally applied pathogen case, the Jacobian for the externally applied pathogen is also given

by (2.3).

1. For the pathogen only steady state, the spectrum of the Jacobian is

$$\sigma(J) = \left\{ r_S - \frac{\beta a}{d}, r_R, -\delta, -d \right\}.$$

Since $0 < r_R \in \sigma(J)$ the steady state is unstable. Hence it is impossible to completely eradicate the aphid population. However, if there are no resistant aphids present (i.e. $R = 0$) then the steady state is stable if, and only if,

$$a > \frac{dr_S}{\beta},$$

that is, provided that sufficient amounts of externally applied pathogen is used.

2. For the resistants and pathogen steady state the spectrum of the Jacobian is

$$\sigma(J) = \left\{ r_S \left(1 - \frac{\alpha_1 K_R}{K_S} \right) - \frac{\beta a}{d}, -r_R, -\delta, -d \right\}$$

and so the steady state is stable if, and only if,

$$r_S \left(1 - \frac{\alpha_1 K_R}{K_S} \right) - \frac{\beta a}{d} < 0. \tag{2.10}$$

Thus if in the absence of the pathogen the resistants are strong competitors (such that $K_S - \alpha_1 K_R < 0$) then the steady state will always be stable and the resistants will always persist. Moreover, if the resistants are weak competitors (such that $K_S - \alpha_1 K_R > 0$) then in the absence of the pathogen the susceptibles may be able to persist, but by applying sufficient amounts of pathogen (such that (2.10) holds) it will be possible to eradicate the susceptible aphid population. Moreover, if

$$a > \frac{dr_S}{\beta} \tag{2.11}$$

then the steady state will be stable and no biologically realistic resistants eliminated steady state will exist.

3. The resistants-eliminated steady state is stable if, and only if,

$$\begin{aligned} 0 &> K_R - \alpha_2 \hat{S} \\ 0 &< a_3 \\ 0 &< a_1 a_2 - a_3 \end{aligned}$$

where

$$\begin{aligned} a_1 &= \frac{r_S}{K_S}\hat{S} + \delta + d \\ a_2 &= \frac{r_S}{K_S}(\delta + d)\hat{S} + \delta(d - \omega\beta\hat{S}) \\ a_3 &= \omega\delta\beta^2\hat{S}\hat{F} + \frac{r_S\delta}{K_S}\hat{S}(d - \omega\beta\hat{S}) \end{aligned}$$

Note that $a_3 > 0$ if $\hat{S} < \frac{d}{\omega\beta}$.

4. For the non-trivial steady state the conditions for stability can be written down using the Routh-Hurwitz conditions for quartics but have no biologically tractable meaning, and so they are not discussed here.

2.2.4 Varying β

An important question to ask is whether if we change the pathogenicity of the fungus that is applied to the aphids, will there be any dynamical differences observed? This is an important question from a control point of view since it may be beneficial to some extent to determine which biotype of aphid will survive.

As a starting point I shall consider the case where $\beta = 0$. This is essentially the Lotka-Volterra competition model. As we have seen, there can be only three types of behaviour where we have two types of competitive exclusion or coexistence. In Figure 2-1 the parameters have been chosen to give coexistence, and the value for a has been chosen to be small, so that the effects of varying β can been seen clearly.

Now using a small value for beta ($\beta = 0.1$) we can see that comparing Figure 2-1 and Figure 2-2 that very little has changed in the behaviour. What this tells us is that if the mycoinsecticide is not virulent enough, then it's a ineffective form of control.

Increasing the value of β a little more causes the total density of healthy aphids to drop (i.e. $S + R$ gets smaller). From a control point of view this is what one would hope for, as reducing the density of healthy aphids reduces the amount of damage caused to the crops. Notice however that in Figure 2-3 although the total density of aphids drops, the actual number of resistant aphids actually increases, and it is the number of susceptible aphids that decrease significantly.

As β increases further (see Figure 2-4) one can see that damped oscillations start to occur. Note that the total number of aphids is still decreasing.

When the value of β becomes sufficiently high (as in Figure 2-5) the steady state loses its stability and we observe unstable oscillatory behaviour. With this type of behaviour it is difficult to predict the population sizes at a given time or in the distant future.

Finally we look at the case where β is very large (see Figure 2-6). In this case β has become sufficiently large that (2.10) is true. Hence, we see competitive exclusion since

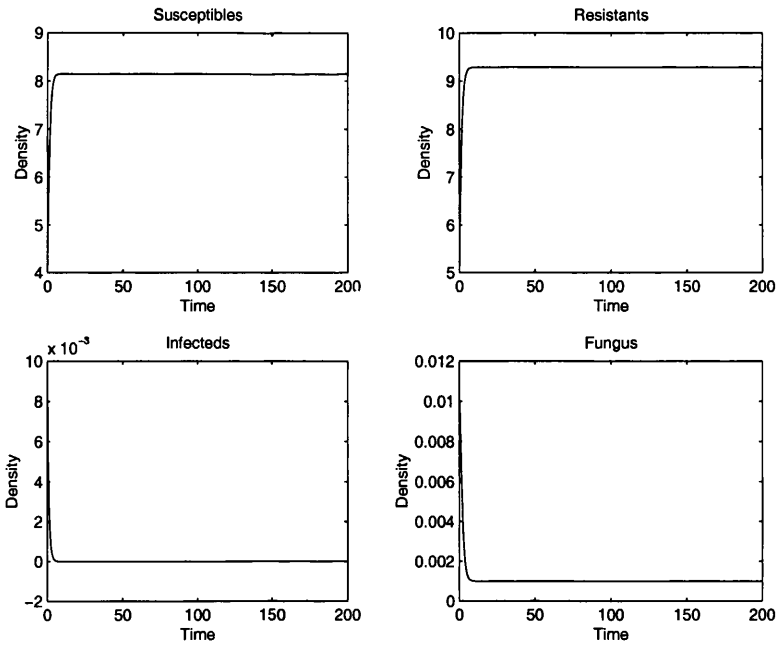


Figure 2-1: Coexistence in the Competition Model $r_S = 1, K_S = 10, \alpha_1 = 0.2, r_R = 0.9, K_R = 10.1, \alpha_2 = 0.1, \delta = 1, a = 0.001, d = 1, \omega = 1$ and $\beta = 0$.

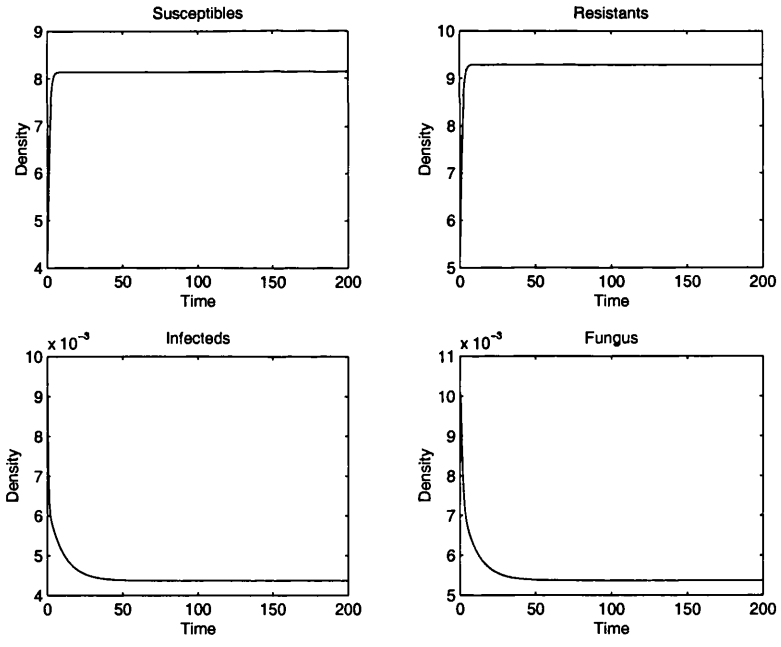


Figure 2-2: Varying β: Small Beta $r_S = 1, K_S = 10, \alpha_1 = 0.2, r_R = 0.9, K_R = 10.1, \alpha_2 = 0.1, \delta = 1, a = 0.001, d = 1, \omega = 1$ and $\beta = 0.1$.

the susceptibles are unable to cope with the highly infectious fungus. This is the best strategy for control in the case where in the absence of the fungal control one observes

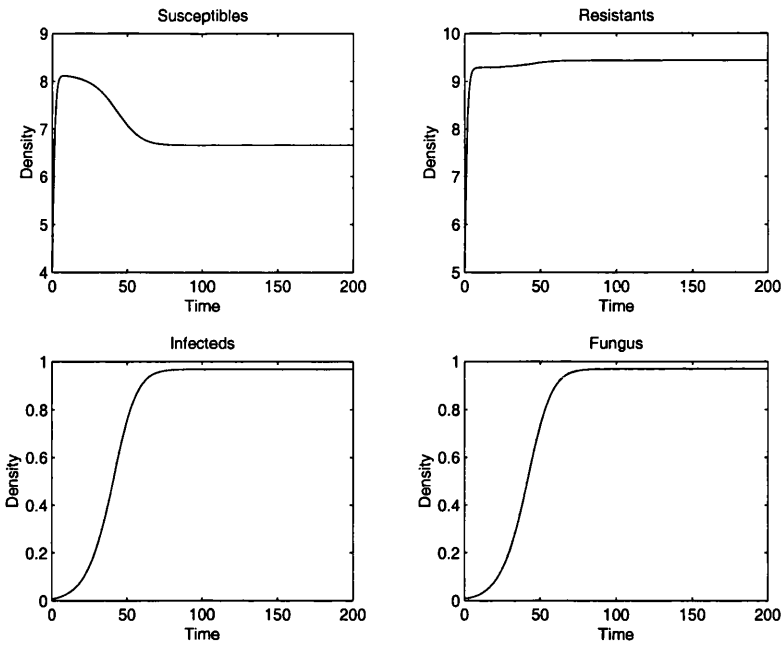


Figure 2-3: Varying β: Moderately Small Beta $r_S = 1, K_S = 10, \alpha_1 = 0.2, r_R = 0.9, K_R = 10.1, \alpha_2 = 0.1, \delta = 1, a = 0.001, d = 1, \omega = 1$ and $\beta = 0.15$.

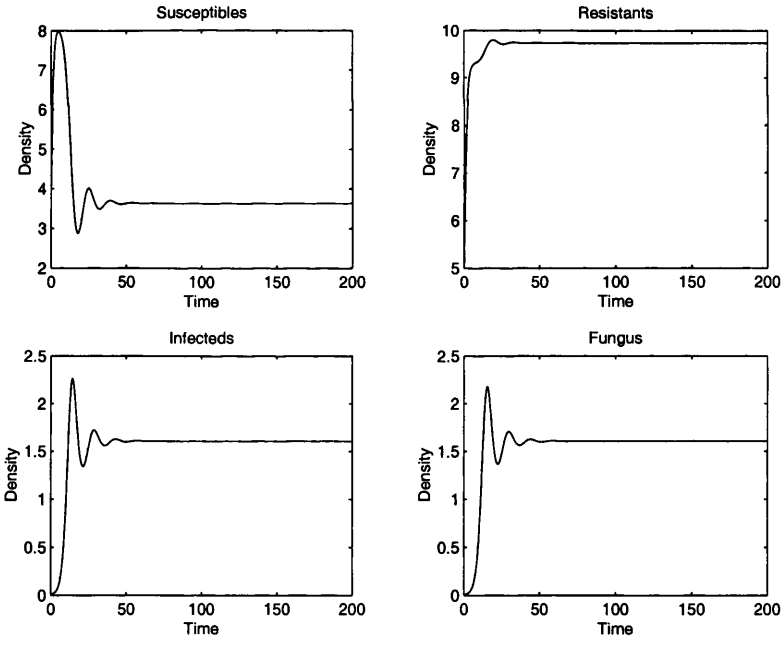


Figure 2-4: Varying β: Moderate Beta $r_S = 1, K_S = 10, \alpha_1 = 0.2, r_R = 0.9, K_R = 10.1, \alpha_2 = 0.1, \delta = 1, a = 0.001, d = 1, \omega = 1$ and $\beta = 0.275$.

coexistence.

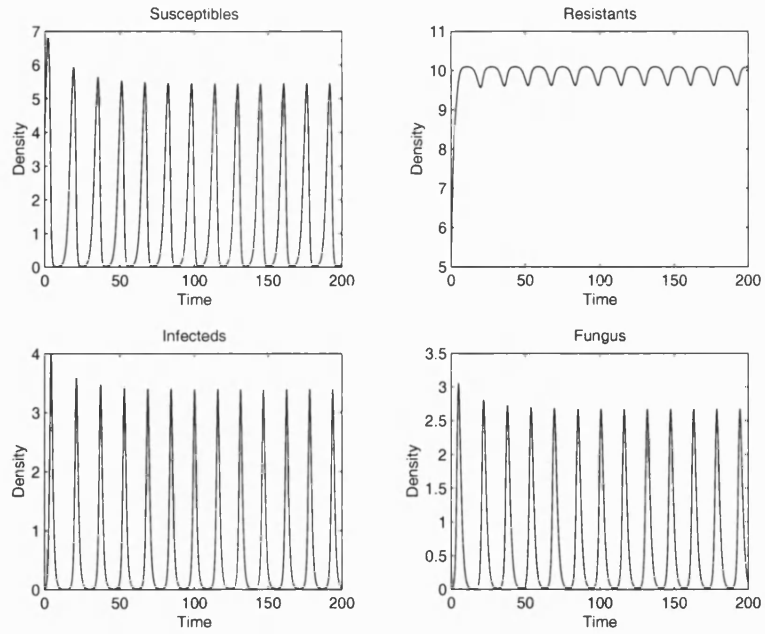


Figure 2-5: Varying β: Moderately High Beta $r_S = 1, K_S = 10, \alpha_1 = 0.2, r_R = 0.9, K_R = 10.1, \alpha_2 = 0.1, \delta = 1, a = 0.001, d = 1, \omega = 1$ and $\beta = 1$.

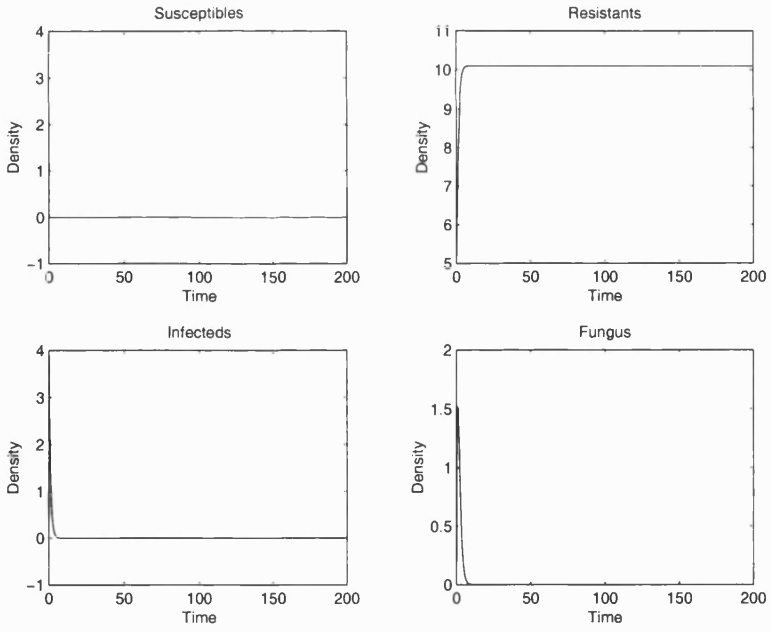


Figure 2-6: Varying β: High Beta $r_S = 1, K_S = 10, \alpha_1 = 0.2, r_R = 0.9, K_R = 10.1, \alpha_2 = 0.1, \delta = 1, a = 0.001, d = 1, \omega = 1$ and $\beta = 800$.

Note that if

$$\beta \gg \frac{dr_S}{a}\left(1 - \frac{\alpha_1 K_R}{K_S}\right) \tag{2.12}$$

then the behaviour of the system will not change in that you reach the same steady state. However, the speed at which the steady state is approached will be increased. This could be important from a costing point of view since it could cost more to produce a highly infective fungus, although it may be desirable to produce a fast-acting control. This type of trade-off would be important in a marketing plan.

2.3 Spatially Extended Continuous-Time Model

An important question to ask is whether spatial effects can influence the dynamics of the aphid interactions. One possible way to do this is to consider a PDE model which has diffusion terms to represent aphid movement and reaction terms to represent the aphid interactions. In the models that I have considered such a model could take the form:

$$\begin{aligned}
\frac{\partial S}{\partial t} &= r_S S\left(1-\frac{S+\alpha_1 R}{K_S}\right) - \beta SF + D_S\frac{\partial^2 S}{\partial x^2} \\
\frac{\partial R}{\partial t} &= r_R R\left(1-\frac{R+\alpha_2 S}{K_R}\right) + D_R\frac{\partial^2 R}{\partial x^2} \\
\frac{\partial I}{\partial t} &= \beta Sf - \delta I + D_I\frac{\partial^2 I}{\partial x^2} \\
\frac{\partial F}{\partial t} &= a - dF + \omega\delta I + D_F\frac{\partial^2 F}{\partial x^2}
\end{aligned}$$

in 1D.

However, there are assumption problems with such a model. For example the above model assumes that space is continuous. However, this may not be true since the sites (i.e. plants) that aphids occupy are not continuously spread especially when dealing with larger plant types in the greenhouses or fields. Therefore perhaps a more realistic approach would be to use an ODE lattice.

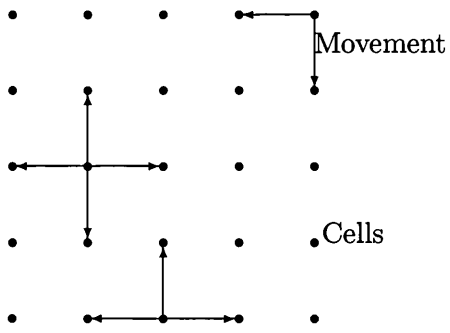


Figure 2-7: ODE Lattice Diagram

The ODE lattice uses the full continuous time model (2.2) for the local dynamics. When the joint density of susceptible and resistant aphids reach some threshold density, K^* say, a certain proportion of each aphid type is able to move to a neighbouring site (see Figure 2-7). The reasoning for such dispersion arises from the crowding response of aphids.

Unfortunately I was unable to derive any analytical results from the automata, however I did carry out an extensive series of numerical simulations which indicated some interesting results. I found that domain size (i.e. the number of cells) did not make a difference to the outcomes of the spatial model, and so I used a 10×10 grid for all simulations. Reflective boundary conditions were also used.

To begin with I looked at the case where no fungal pathogen was present and began the simulation at opposite corners of the array (see Figure 2-8). We can see that since

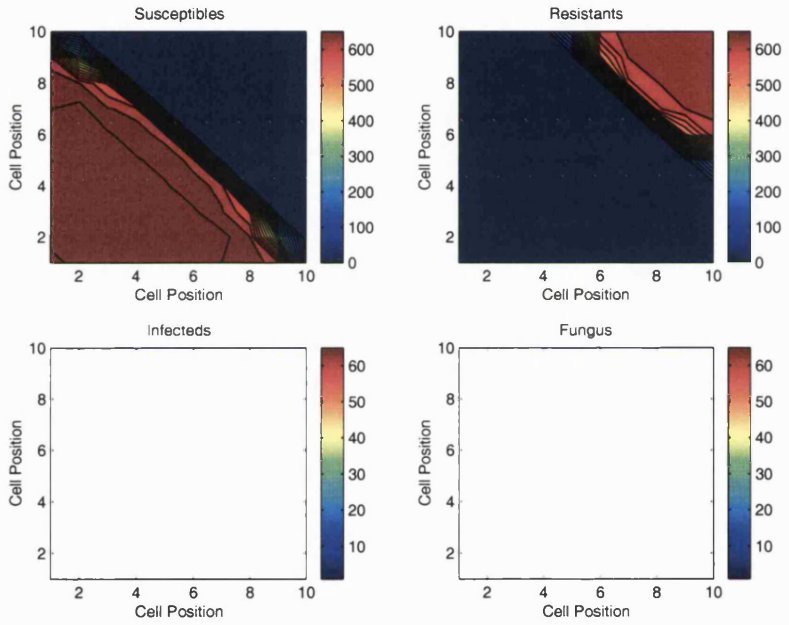


Figure 2-8: No Fungal Pathogen: $r_S = 1, \alpha_1 = 0.8, K_S = 700, r_R = 0.7, \alpha_2 = 0.1, K_R = 650$ and $K^* = 600$.

$r_S > r_R$ the susceptibles can invade more quickly. This makes sense because as both aphid strains reproduce at a site only a small number of these types move to a new site as a response to crowding. Therefore as $r_S > r_R$ we know that the susceptibles reproduce more quickly at low density and therefore reach thier threshold density more quickly and thus spread throughout the domain more quickly. When the simulation is run for a longer period of time, we see that the populations settle down to a steady state distribution as predicted by the temporal model (see Figure 2-9).

Now what difference does the inclusion of the fungal pathogen have? I ran the same sim-

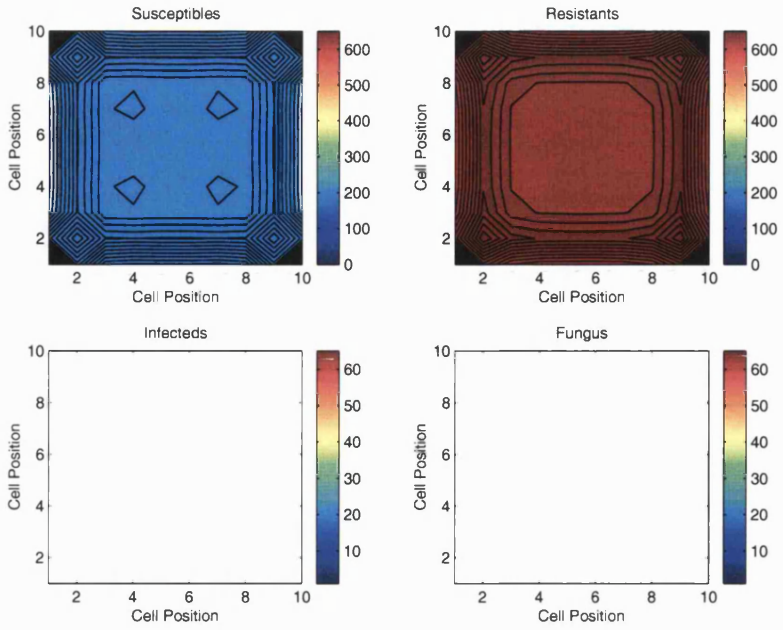


Figure 2-9: No Fungal Pathogen: $r_S = 1, \alpha_1 = 0.8, K_S = 700, r_R = 0.7, \alpha_2 = 0.1, K_R = 650$ and $K^* = 600$.

ulation as above but this time included the parameters of the fungal pathogen and the difference was clear to see (see Figure 2-10). We see that the fungal pathogen suppresses

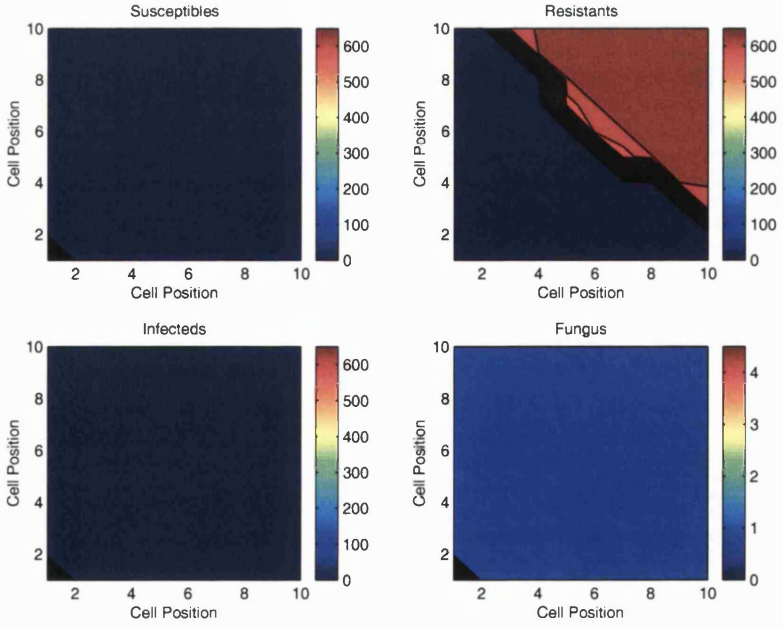


Figure 2-10: With Fungal Pathogen: $r_S = 1, \alpha_1 = 0.8, K_S = 700, r_R = 0.7, \alpha_2 = 0.1, K_R = 650, \delta = 0.3, a = 1, d = 1.5, \omega = 0.05$ and $K^* = 600$.

the susceptible aphid movement. This is because the fungus significantly reduces the steady state value of susceptibles to lower than the threshold density. Therefore the susceptibles do not move because there is no significant crowding. However, since the resistants are not affected by the pathogen they are free to move about the domain. When the simulation is run a little longer (see Figure 2-11) we can see that the resistants

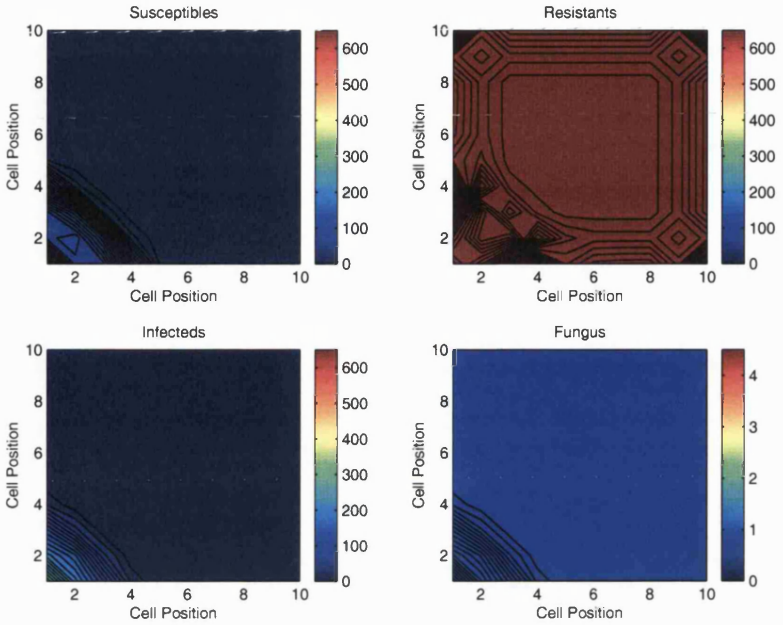


Figure 2-11: With Fungal Pathogen: $r_S = 1, \alpha_1 = 0.8, K_S = 700, r_R = 0.7, \alpha_2 = 0.1, K_R = 650, \delta = 0.3, a = 1, d = 1.5, \omega = 0.05$ and $K^* = 600$.

begin to approach their equilibrium distribution. However, in the corner where the two biotypes begin to mix we see that the previously stationary susceptible aphids begin to move. This is due to the mixing effect and thus the susceptibles begin to respond to crowding provided by the resistant strain. Note that although the susceptibles can now move, their density is still relatively low. Eventually this simulation leads to an equilibrium distribution where the resistants are close to their carrying capacity and the susceptibles do survive but at a low level, which is consistent with the temporal model prediction.

One might ask whether spatial effects can alter the behaviour of a temporal model. For example, can one observe any spatial patterning or patchiness? Unfortunately with my numerical studies I did not observe any such behaviour. However, when exploring the parameter domain where it is possible to observe local stability of equilibria I found that when using a random distribution of starting values the resulting equilibrium distribution always favoured the resistant aphids.

2.4 Conclusions & Discussion

As the analysis of the models show, the presence of resistant insects can have negative effects on the control strategy. When using a single application of fungal pathogen, it is impossible to eradicate the aphids even if there are no resistant aphids present. However, using a constant spraying strategy, this is no longer the case. If the resistants are absent, then eradication is possible if sufficient amounts of the pathogen are applied. However, if the resistants are present, then eradication is impossible.

The effects of competition between the two aphid types does not help with the total species eradication. However, it does play an important part in the eradication of one of the species. If the resistants are strong competitors and the susceptibles are weak competitors then the susceptibles will always be eliminated, independent of the spraying strategy. But, if the competition roles are reversed then the spraying strategy plays a part as to which of the two aphid species persists. In both the spraying strategy cases, the susceptibles must be able to sustain their competitive advantage over the resistants in the presence of the pathogen. However, by increasing the amount of spray applied it is possible to destabilise the steady state. Clearly this is not the case with the single application strategy.

What is clear from this analysis, is that if the sufficiently large amounts of the fungal pathogen is applied, then the susceptible insects can be eradicated, but will be replaced by resistant insects.

When both types of aphid coexist, the susceptible aphid levels decrease as the amount of pathogen increases. But since $R^* = K_R - \alpha_2 S^*$, the resistant aphid levels must therefore increase. This is intuitively obvious since an increase in the pathogen levels must decrease the susceptible numbers, and therefore decrease the amount of competition exerted on the resistants, and so the resistant numbers increase.

As shown in Section 2.2.4, the non-trivial steady state may be destabilised, so that oscillatory behaviour will be observed. The numerical simulations show that if the pathogen is not administered carefully, then the result may be insect outbreaks, making control of the pests difficult to assess.

From the spatially extended model, we see that the fungal pathogen may reduce the spread of the susceptible insects since the pathogen reduces the susceptible insect numbers, and hence there is little crowding, and so the susceptible insects do not move. However, if resistant insects are present (which are not affected by the pathogen), then when both species share a site the numbers may cause sufficient crowding so that the susceptible insects disperse.

Chapter 3

Time-Dependent Spraying Strategies

3.1 Motivation and Background

3.1.1 Chapter Outline

This chapter uses the continuous-time model developed in Chapter 2 to investigate the scenario when the control is not applied continually, but instead is applied in pulses. I consider two forms of control, firstly a chemical insecticide and then the fungal pathogen, and in each case, discuss when resistance has developed or not. I also develop the notion of a minimal spraying strategy and derive conditions for the pest insect to be eradicated.

3.1.2 Motivation

In Chapter 2 it is assumed that the control is applied to the environment at constant rate, independent of the control levels. However, this may not be very realistic. Perhaps a more realistic assumption is that the control is applied at regular or irregular intervals. Chemical and pathogen controls are mostly applied either by a foliar spray, or, in the case of some chemical insecticides, by applying the control to the soil and relying on the plant to take up the control as it feeds, thus providing protection as the plant grows. The models presented in this thesis are only suitable for foliar applications, where typically the controls are applied in pulses.
This idea of a pulsed spray forms the basis for the topic of this chapter.

3.1.3 Background Reading

In Shulgin, Stone & Agur (1998) the authors consider a simple SIR model used to model a measles epidemic. The SIR model equations are

$$\begin{aligned} \frac{dS}{dt} &= m - (\beta I + m)S \\ \frac{dI}{dt} &= \beta IS - (m + g)I \\ \frac{dR}{dt} &= gI - mR \end{aligned}$$

where S, I and R are the densities of susceptibles, infected and removed hosts at time t, m is the birth and death rate, β is the contact rate, g is the removal rate (i.e. the rate at which the infected become immune to the disease), and hence $1/g$ is the mean infectious period and the total population size has been normalised to unity:

$$S(t) + I(t) + R(t) = 1.$$

Further information about the model assumptions and its analysis can be found in Anderson & May (1992).

The authors then considered two different types of vaccination strategies. Firstly they considered a constant vaccination strategy, by vaccinating a proportion, p, of all newborn so that the susceptible equation becomes

$$\frac{dS}{dt} = (1-p)m - (\beta I + m)S.$$

The second strategy is a pulse strategy where a proportion p of the entire susceptible population is vaccinated in a single pulse, every T years. The underlying principle is to apply vaccination pulses frequently enough so as to prevent the infectious population from ever growing, i.e., by maintaining $dI/dt < 0$ for all time. Such a strategy will ensure that $I(t)$ is a decreasing function of time, and the infectious population will eventually dwindle to zero. For this strategy, the susceptible equation becomes

$$\frac{dS}{dt} = m - (\beta I + m)S - p\sum_{n=0}^{\infty} S(nT^-)\delta(t - nT).$$

where

$$S(nT^-) = \lim_{\varepsilon \to 0} S(nT - \varepsilon), \ \varepsilon > 0.$$

Two types of analysis are carried out. The first revolves about the derivation of a stroboscopic (discrete time) map, under the assumption that $I(t) = 0$ for all $t \geq 0$, which is motivated by the fact that $I^* = 0$ is an equilibrium solution for $I(t)$. Stability

analysis is then carried out on the map to determine conditions for which the infection can be eradicated. The second uses Floquet Theory (Iooss & Joseph 1980) to determine a time, $T_{\max}$, which is the maximum allowable period of the pulse, such that the 'infection free' equilibrium is stable. For this theory to be used, the authors rely on the calculation of Floquet multipliers, which may be difficult to compute for more complicated models.

The main pitfall of this analysis is that it relies on the time-dependent solution of an ODE or system of ODEs. For many models this is not possible, even under severe assumptions. However, it is possible for some simple models, and of course numerical simulations are simple to run.

Other examples of periodically pulsed systems can be found. For example in Funasaki & Kot (1993) the authors consider a model for a chemostat (a common piece of laboratory apparatus used to culture microorganisms) with predator-prey dynamics and a periodically pulsed substrate. Stroboscopic maps are then derived and analysed. In Sabin & Summers (1992) the authors consider a periodically forced Lotka-Volterra predator-prey model with logistic prey growth, where the forcing term enters via a periodically varying intrinsic growth rate in the prey. Numerical simulations are then carried out, as well as graphing Poincaré maps (the result of graphing stroboscopic maps). In both of these papers the authors found that simple cycles may give way to chaos in a cascade of period-doubling bifurcations.

In Sherratt (1995), the author considers the possibility of Turing bifurcations when the parameter values (both diffusion coefficients and kinetic parameters) oscillate in time between two sets of constant values, with a period that is either very short or very long compared to the time scale of the growth and predation kinetics. In particular, the model investigated is

$$\begin{aligned} \frac{\partial u}{\partial t} &= D_u(t)\nabla^2 u + f(u,v,t) \\ \frac{\partial v}{\partial t} &= D_v(t)\nabla^2 v + g(u,v,t) \end{aligned}$$

where on $nT < t < (n+1/2)T$

$$D_u(t) \equiv D_{u,1}, \quad D_v(t) \equiv D_{v,1}, \quad f(u,v,t) \equiv f_1(u,v), \quad g(u,v,t) \equiv g_1(u,v)$$

and on $(n+1/2)T < t < (n+1)T$

$$D_u(t) \equiv D_{u,2}, \quad D_v(t) \equiv D_{v,2}, \quad f(u,v,t) \equiv f_2(u,v), \quad g(u,v,t) \equiv g_2(u,v)$$

and where T is the period and $n \in \mathbb{N}$.

The author assumes that f_i and g_i are such that the system has a spatially homogeneous solution $(u,v) = (u_s(t), v_s(t))$ that is periodic in time with the same period T as the

model parameters. As pointed out in the paper, one intuitively expects such a solution to exist for ecologically realistic models, but conditions on f_i and g_i for such a solution to exist are not known by the author, nor by myself.
Although the author is mainly concerned with diffusion-driven instability as $T \to 0$ and $T \to \infty$, conditions are derived for the spatially homogeneous solution to be stable.
In the limit as $T \to 0$, the spatially homogeneous solution is stable if the eigenvalues of the matrix M have negative real part, where

$$M = \begin{bmatrix} \bar{a} & \bar{b} \\ \bar{c} & \bar{d} \end{bmatrix}$$

with

$$\begin{aligned}
\bar{a} &= 1/2\left(\left.\frac{\partial f_1}{\partial u}\right|_{(u_0,v_0)} + \left.\frac{\partial f_2}{\partial u}\right|_{(u_0,v_0)}\right) \\
\bar{b} &= 1/2\left(\left.\frac{\partial f_1}{\partial v}\right|_{(u_0,v_0)} + \left.\frac{\partial f_2}{\partial v}\right|_{(u_0,v_0)}\right) \\
\bar{c} &= 1/2\left(\left.\frac{\partial g_1}{\partial u}\right|_{(u_0,v_0)} + \left.\frac{\partial g_2}{\partial u}\right|_{(u_0,v_0)}\right) \\
\bar{d} &= 1/2\left(\left.\frac{\partial g_1}{\partial v}\right|_{(u_0,v_0)} + \left.\frac{\partial g_2}{\partial v}\right|_{(u_0,v_0)}\right)
\end{aligned}$$

and (u_0, v_0) is a solution of

$$f_1(u_0, v_0) + f_2(u_0, v_0) = g_1(u_0, v_0) + g_2(u_0, v_0) = 0.$$

The interpretation of (u_0, v_0) can be thought of as the average equilibrium values over the period. Hence the stability condition is reduced to

$$\bar{a} + \bar{d} < 0 \quad \text{and} \quad \bar{a}\bar{d} - \bar{b}\bar{c} > 0$$

by the Routh-Hurwitz conditions for quadratics (see Appendix A.2). The values $\bar{a}$, $\bar{b}$, $\bar{c}$ and $\bar{d}$ can be thought of as the average values of the Jacobian over the period.
In the limit as $T \to \infty$ an additional assumption was made on the kinetics, that for both $i = 1$ and $i = 2$, $f_i(u, v)$ and $g_i(u, v)$ have a unique steady state in the first positive quadrant, that is both globally attracting in this quadrant and also linearly stable (this second assumption rules out the possibility of neutral stability). Let these steady states be denoted by $(u_{s,i}, v_{s,i})$ for $i = 1, 2$.
Now for $i = 1, 2$ let

$$a_i = \left.\frac{\partial f_i}{\partial u}\right|_{u_{s,i},v_{s,i}} \quad b_i = \left.\frac{\partial f_i}{\partial v}\right|_{u_{s,i},v_{s,i}} \quad c_i = \left.\frac{\partial g_i}{\partial u}\right|_{u_{s,i},v_{s,i}} \quad d_i = \left.\frac{\partial g_i}{\partial v}\right|_{u_{s,i},v_{s,i}},$$

and let $\tau_i = a_i + d_i$ and $\Delta_i = a_i d_i - b_i c_i$. Then the author shows that the homogeneous solution is stable if

$$\begin{aligned}
&\tau_2(2\tau_1 + \tau_2) + 4\Delta_1 > 0 \\
&\tau_1(2\tau_2 + \tau_1) + 4\Delta_2 > 0 \\
&(\tau_2\Delta_1 + \tau_1\Delta_2)(\tau_1 + \tau_2) + (\delta_1 - \Delta_2)^2 > 0 \\
&\tau_1 + \tau_2 < 0.
\end{aligned}$$

I now follow this analysis for the system below.

3.2 The Time-Dependent Spraying Strategy Model

3.2.1 Model Formulation

Consider Model 2.2 where $S(t), R(t), I(t)$ and $F(t)$ are the densities of susceptibles, resistants, infecteds and free-living fungal spores at time t. For the purposes of this chapter, I will redefine $F(t)$ as the density of control (either a chemical insecticide or fungal pathogen) at time t. I also remove the constant spraying strategy, a, and include a time dependent spraying strategy by considering a sequence of times $\{t_0, t_1, t_2, \dots\}$ where the control is sprayed on the foliage. At each time t_i, a set amount, a, of control, F, is applied. Moreover, I do not explicitly model the infected class, and assume that the per capita rate at which the susceptibles become infected/inoculated is directly proportional to the density of control in the environment (i.e. the mass-action law). Hence for $t \in (t_n^+, t_{n+1}^-)$, Model 2.2 becomes:-

$$\frac{dS}{dt} = r_S S\left(1 - \frac{S + \alpha_1 R}{K_S}\right) - \beta SF \tag{3.1a}$$

$$\frac{dR}{dt} = r_R R\left(1 - \frac{R + \alpha_2 S}{K_R}\right) \tag{3.1b}$$

$$\frac{dF}{dt} = \omega\beta SF - dF \tag{3.1c}$$

with the conditions

$$S(t_n^-) = S(t_n^+) = S_n \tag{3.1d}$$

$$R(t_n^-) = R(t_n^+) = R_n \tag{3.1e}$$

$$F(t_n^+) = F(t_n^-) + a \tag{3.1f}$$

$$F(t_n^-) = F_n \tag{3.1g}$$

where S_n, R_n and F_n are the densities of susceptibles, resistants and control remaining at the end of the previous interval.

To analyse Model 3.1 I consider two cases. The first is when the control is a chemical insecticide, and the second when the control is a fungal pathogen. I then compare the two results.

3.2.2 Chemical Insecticide

The main difference between most chemical insecticides and fungal pathogens is that when the host insect is inoculated with the insecticide, the poison kills the host (if given a large enough dose) and remains within the host. Thus there is no possibility of secondary infections, and hence $\omega = 0$.
Let us first consider the case when no resistance develops.

No Resistance

With $R(t) \equiv 0$ and $\omega = 0$, Model 3.1 becomes

$$\frac{dS}{dt} = r_S S\left(1 - \frac{S}{K_S}\right) - \beta SF \tag{3.2a}$$

$$\frac{dF}{dt} = -dF \tag{3.2b}$$

for $t \in (t_n^+, t_{n+1}^-)$, with

$$S(t_n^-) = S(t_n^+) = S_n \tag{3.2c}$$

$$F(t_n^+) = a + F(t_n^-) = a + F_n. \tag{3.2d}$$

If there is only a single application of chemical insecticide at $t_0 = 0$ then the long-term behaviour is simple to determine. The trivial steady state $(S^*, F^*) = (0, 0)$ is unstable, whereas the steady state $(S^*, F^*) = (K_S, 0)$ is always stable. In other words, if there is one initial application of the chemical insecticide, the chemical will simply decay in the environment and the susceptible insects will go to their carrying capacity. Therefore, it is impossible to eradicate the insects with a single application of chemical.
Can we control the insect population using a time-dependent spraying strategy? For $t \in (t_n^+, t_{n+1}^-)$ we can integrate (3.2b) to get

$$F(t) = F(t_n^+)e^{-d(t-t_n^+)}.$$

Thus,

$$\begin{aligned} F_{n+1} &= F(t_{n+1}^-) \\ &= F(t_n^+)e^{-d(t_{n+1}^- - t_n^+)} \\ &= (a + F(t_n^-))e^{-d(t_{n+1}^- - t_n^+)} \\ &= (a + F_n)e^{-d(t_{n+1}^- - t_n^+)}. \end{aligned}$$

Now we can rearrange (3.2a) to get the Bernoulli equation

$$\frac{dS}{dt} + (\beta F(t) - r_S)S = -\frac{r_S}{K_S}S^2.$$

So substituting $V = S^{-1}$ we get the linear equation

$$\frac{dV}{dt} + (r_S - \beta F(t))V = \frac{r_S}{K_S}.$$

Thus, introducing the integrating factor

$$\hat{E}(t) = \exp\left(r_S t + \frac{\beta}{d}F(t_n^+)e^{-d(t-t_n^+)}\right),$$

we get

$$\frac{d}{dt}\left(\hat{E}V\right) = \frac{r_S}{K_S}\hat{E},$$

and integrating from t_n^+ to t we have

$$\hat{E}(t)V(t) - \hat{E}(t_n^+)V(t_n^+) = \frac{r_S}{K_S}\int_{t_n^+}^{t} \hat{E}(u)\,du.$$

Hence

$$S(t) = \frac{K_S S(t_n^+)\hat{E}(t)}{r_S S(t_n^+)\int_{t_n^+}^{t}\hat{E}(u)\,du + K_S\hat{E}(t_n^+)}. \tag{3.3}$$

Finally setting $t = t_{n+1}^-$ in (3.3) we get the system of discrete maps

$$S_{n+1} = \frac{K_S S_n \hat{E}(t_{n+1}^-)}{r_S S_n \int_{t_n^+}^{t_{n+1}^-}\hat{E}(u)\,du + K_S\hat{E}(t_n^+)} \tag{3.4a}$$

$$F_{n+1} = (a + F_n)e^{-d(t_{n+1}^- - t_n^+)} \tag{3.4b}$$

where

$$\hat{E}(t) := \exp\left\{\frac{1}{d}\left(r_S dt + \beta(a+F_n)e^{-d(t-t_n^+)}\right)\right\}. \tag{3.4c}$$

So that the reader can easily follow the analysis of Model 3.4, I have broken the results down into a series of lemmas.

Lemma 3.2.1. *If there exists a positive steady state, the time between successive sprays, T_n, must satisfy*

$$T_n := t_{n+1}^- - t_n^+ > \frac{\beta a}{dr_S}, \tag{3.5}$$

where T_n must be constant and independent of n.

Proof. At steady state we have that

$$S_{n+1} = S_n = S^*$$
$$F_{n+1} = F_n = F^*$$

and so

$$F^* = \frac{ae^{-dT_n}}{1-e^{-dT_n}} > 0. \tag{3.6}$$

Now, (3.4a) gives that either $S^* = 0$ (the trivial steady state) or

$$r_S S^* \int_{t_n^+}^{t_{n+1}^-} \hat{E}(u)\,du = K_S\left(\hat{E}(t_{n+1}^-) - \hat{E}(t_n^+)\right).$$

But $\hat{E}(t) > 0\ \forall t$ and thus

$$\begin{aligned}
\exists S^* > 0 &\Leftrightarrow \hat{E}(t_{n+1}^-) - \hat{E}(t_n^+) > 0 \\
&\Leftrightarrow r_S dt_{n+1}^- + \beta(a+F^*)e^{-dT_n} > r_S dt_n^+ + \beta(a+F^*) \\
&\Leftrightarrow r_S dT_n > \beta(a+F^*)(1-e^{-dT_n}) \\
&\Leftrightarrow T_n > \frac{\beta a}{dr_S}
\end{aligned}$$

□

Lemma 3.2.2. *When using a periodic spraying strategy, $t_n = nh$, for some $h > 0$, the resulting system of discrete equations* (3.4) *are autonomous, and are given by*

$$S_{n+1} = \frac{K_S S_n \exp\left(r_S h + \frac{\beta}{d}(a+F_n)e^{-dh}\right)}{r_S S_n \int_0^h \exp\left(r_S v + \frac{\beta}{d}(a+F_n)e^{-dv}\right)dv + K_S e^{\frac{\beta}{d}(a+F_n)}} \tag{3.7a}$$

$$F_{n+1} = (a+F_n)e^{-dh}. \tag{3.7b}$$

Proof. With the periodic spraying strategy, $t_n = nh$, the system of discrete equations (3.4) become (dropping the upper and lower limits as they can be neglected from the following calculations)

$$\begin{aligned} S_{n+1} &= \frac{K_S S_n \hat{E}((n+1)h)}{r_S S_n \int_{nh}^{(n+1)h} \hat{E}(u)\,du + K_S \hat{E}(nh)} \\ F_{n+1} &= (a + F_n)e^{-dh} \end{aligned}$$

where

$$\hat{E}(t) = \exp\left\{\frac{1}{d}\left(r_S dt + \beta(a+F_n)e^{-d(t-nh)}\right)\right\}.$$

Thus,

$$\begin{aligned} \hat{E}((n+1)h) &= \exp(r_S nh)\exp\left(r_S h + \frac{\beta}{d}(a+F_n)e^{-dh}\right) \\ \hat{E}(nh) &= \exp(r_S nh)\exp\left(\frac{\beta}{d}(a+F_n)\right) \end{aligned}$$

and

$$\begin{aligned} \int_{nh}^{(n+1)h} \hat{E}(u)\,du &= \int_0^h \hat{E}(v+nh)\,dv \\ &= \exp(r_S nh)\int_0^h \exp\left(r_S v + \frac{\beta}{d}(a+F_n)e^{-dv}\right)\,dv. \end{aligned}$$

Then substituting into our discrete equation system we get (3.7). Clearly (3.7) is an autonomous system. □

Lemma 3.2.3. *The discrete system (3.7) admits two steady states and their stability depends on the transcritical bifurcation point $h^* = \frac{\beta a}{dr_S}$. The trivial steady state, $(S^*, F^*) = (0, \tilde{F})$ (where $\tilde{F} > 0$) is stable if, and only if, $h < h^*$. The non-trivial steady state, $(S^*, F^*) = (\tilde{S}, \tilde{F})$ (where $\tilde{S} > 0$) is stable if, and only if, $h > h^*$.*

Proof. Now at steady state we set $S_{n+1} = S_n = S^*$ and $F_{n+1} = F_n = F^*$. Now substituting this in to our system (3.7) we immediately see that

$$F^* = \frac{ae^{-dh}}{1-e^{-dh}} =: \tilde{F},$$

and

$$S^* = 0 \text{ or } S^* = \frac{A^* - C^*}{B^*} =: \tilde{S},$$

where

$$\begin{aligned} A^* &= K_S \exp\left\{ r_S h + \frac{\beta}{d}(a + F^*)e^{-dh} \right\} > 0 \\ B^* &= r_S \int_0^h \exp\left(r_S v + \frac{\beta}{d}(a + F^*)e^{-dv} \right) dv > 0 \\ C^* &= K_S e^{\frac{\beta}{d}(a+F^*)} > 0. \end{aligned}$$

Now clearly $\tilde{F} > 0$ for all parameter values and by Lemma 3.2.1 we have that $\tilde{S} > 0$ if, and only if, $h > h^*$. Thus the two steady states are

$$(S^*, F^*) = (0, \tilde{F}), (\tilde{S}, \tilde{F}).$$

The stability of these steady states depends on the magnitude of the eigenvalues of the Jacobian being less than unity. Now

$$\begin{aligned} \frac{\partial F_{n+1}}{\partial F_n}(S^*, F^*) &= e^{-dh} < 1 \text{ and} \\ \frac{\partial F_{n+1}}{\partial S_n}(S^*, F^*) &= 0. \end{aligned}$$

Hence, the steady states are stable if, and only if,

$$\left| \frac{\partial S_{n+1}}{\partial S_n}(S^*, F^*) \right| < 1.$$

Now,

$$\left| \frac{\partial S_{n+1}}{\partial S_n}(S^*, F^*) \right| = \frac{A^* C^*}{(B^* S^* + C^*)^2},$$

and thus $S^* = 0$ is stable if, and only if, $A^*/C^* < 1$. Now,

$$\begin{aligned} \frac{A^*}{C^*} < 1 &\Leftrightarrow r_S h + \frac{\beta}{d}(a + F^*)e^{-dh} < \frac{\beta}{d}(a + F^*) \\ &\Leftrightarrow r_S h + \frac{\beta}{d}F^* < \frac{\beta}{d}(a + F^*) \\ &\Leftrightarrow h < \frac{\beta a}{d r_S} = h^*. \end{aligned}$$

Also, $S^* = \tilde{S}$ is stable if, and only if, $C^*/A^* < 1$, which is equivalent to $h > h^*$. □

Lemma 3.2.3 is verified numerically by Figure 3-1. It is clear that with a single application of chemical insecticide, the aphid population begins to decline at first, but then as the insecticide decays, the population levels begin to rise, and eventually approach the environmental carrying capacity. In the case where $h > h^*$, the period between

sprays is too long to eradicate the aphid population. However, since the insecticide is repeatedly topped-up, it never completely decays, and so the aphid population is maintained at a lower level than the carrying capacity (this is shown in Lemma 3.2.4). In the case where $h < h^*$ the time between successive sprays is sufficiently small so that the insecticide level is maintained high enough to eradicate the aphid population (recall that $F^* = F^*(h)$ and F^* is a strictly decreasing function of h).

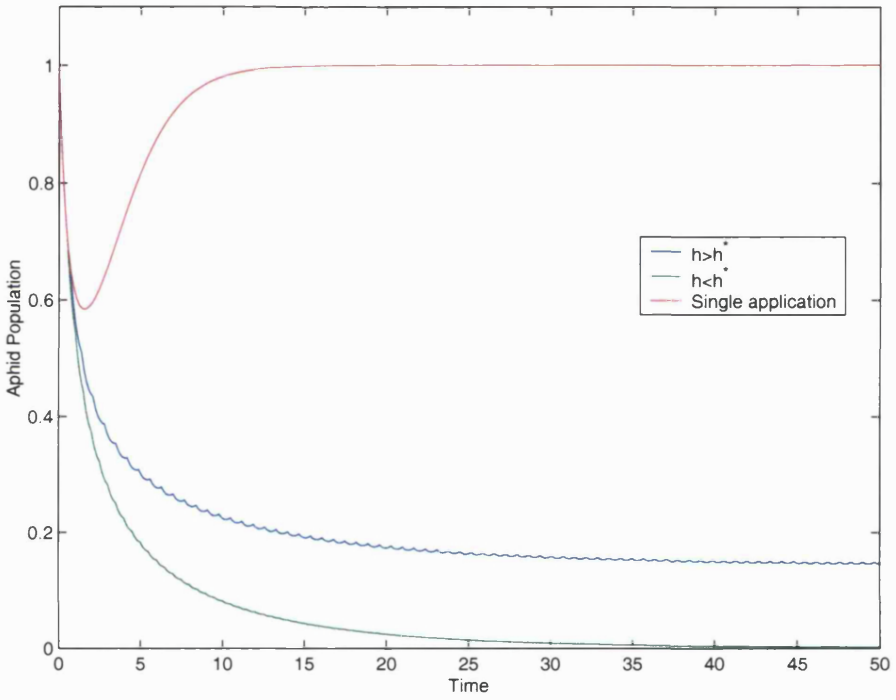


Figure 3-1: Three Spraying Strategies. The parameter values are $r_S = 0.5, K_S = 1, \beta = 1, d = 1, a = 0.3$ and hence $h^* = 0.6$. The case where $h > h^*$ has $h = 0.7$ and where $h < h^*$ has $h = 0.5$. The simulations use the system of differential equations (3.2) and a periodic spraying strategy $t_n = nh$, $h > 0$.

Lemma 3.2.4. *$S^*(h^*) = 0$ and $S^*(h) \to K_S$ as $h \to \infty$.*

Proof. Recall that

$$S^*(h) = \frac{K_S \left\{ e^{r_S h + \frac{\beta}{d} F^*} - e^{\frac{\beta}{d}(a+F^*)} \right\}}{r_S \int_0^h e^{r_S v + \frac{\beta}{d}(a+F^*)e^{-dv}} \, dv}.$$

Then clearly we have that $S^*(h^*) = 0$ since

$$\begin{aligned} e^{r_S h^* + \frac{\beta}{d} F^*} - e^{\frac{\beta}{d}(a+F^*)} &= e^{\frac{\beta}{d} F^*} (e^{r_S h^*} - e^{\frac{\beta a}{d}}) \\ &= 0. \end{aligned}$$

Now $F^* \to 0$ as $h \to \infty$, so

$$\begin{aligned} e^{r_S h + \frac{\beta}{d}F^*} &\sim e^{r_S h} \\ e^{\frac{\beta}{d}(a+F^*)} &\sim e^{\frac{\beta a}{d}} \\ r_S \int_0^h e^{r_S v + \frac{\beta}{d}(a+F^*)e^{-dv}}\, dv &\sim e^{r_S h}. \end{aligned}$$

Therefore,

$$\begin{aligned} S^*(h) &\sim K_S \frac{e^{r_S h} - e^{\frac{\beta a}{d}}}{e^{r_S h}} \\ &= K_S \left(1 - e^{\frac{\beta a}{d} - r_S h}\right) \\ &\to K_S \text{ as } h \to \infty. \end{aligned}$$

□

I have shown that using a periodic spraying strategy it is possible to eradicate the aphid population if the spraying times are sufficiently close together ($h < h^*$), whereas a single application cannot eradicate the pests. In terms of cost effectiveness it may be beneficial to spray as little as possible, therefore increasing the effectiveness of the chemical insecticide (i.e. increasing $1/d$ and β), means that the time between sprays can be decreased.

Notice that h^* does not depend on environmental carrying capacity, K_S. This is due to the fact that the carrying capacity can be scaled out of the equations by making the substitution $s = S/K_S$. Hence, h^* is independent of the carrying capacity.

So far I have shown that if $h < h^*$ then the aphid population can be eradicated. However, can we choose a series spraying times such that we achieve eradication, and do it in a minimalist way?

Consider an interval (t_n^+, t_{n+1}^-). When the chemical insecticide is applied, the aphid population begins to decline (see Figure 3-2), if enough is applied . When the insecticide has sufficiently decayed, the insect population then begins to grow. Thus the idea of the minimal spraying strategy is to pick the spraying times according to the points where growth of the of the insect population is zero.

Lemma 3.2.5. *For $t > t_n^+$ the minimal spraying time is given by*

$$\left(r_S - \beta(a + F_n)e^{-d(t-t_n^+)}\right)\left(r_S S_n \int_{t_n^+}^t \hat{E}(u)\, du + K_S \hat{E}(t_n^+)\right) - r_S S_n \hat{E}(t) = 0. \quad (3.8)$$

Proof. Recall that the governing equation is

$$S(t) = \frac{K_S S_n \hat{E}(t)}{r_S S_n \int_{t_n^+}^t \hat{E}(u)\, du + K_S \hat{E}(t_n^+)}. \quad (3.9)$$

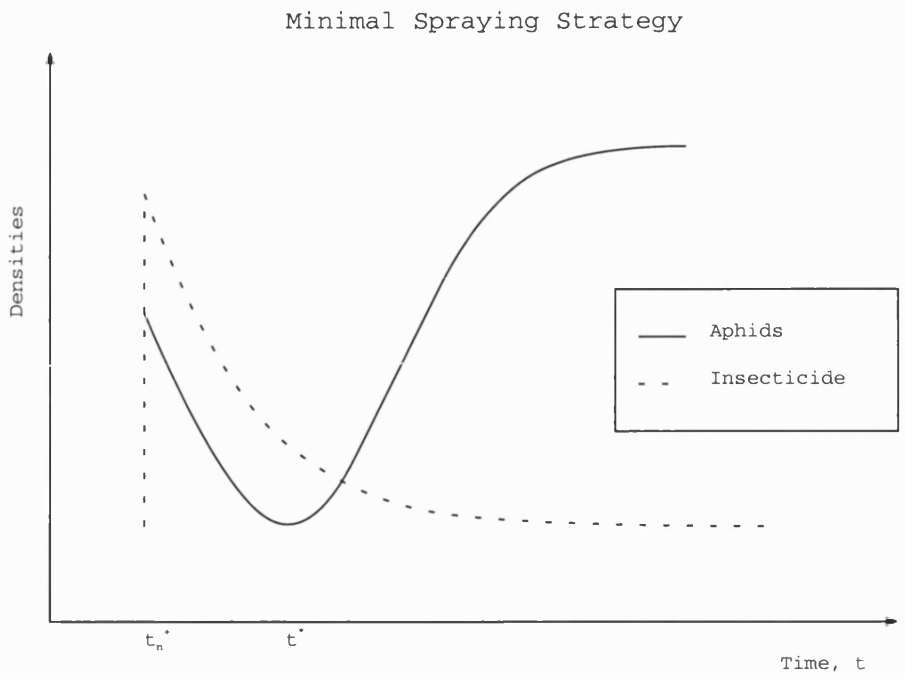


Figure 3-2: Sketch showing the minimal time to spray.

Now the minimal spraying time is simply given by the time $t > t_n^+$ for which $\frac{dS}{dt} = 0$. Thus by differentiating (3.9) and rearranging we get (3.8).
Now, for $t > t_n^+$ we have

$$\begin{aligned} \frac{dS}{dt} &= r_S S\left(1 - \frac{S}{K_S}\right) - \beta SF \\ &= r_S S - \frac{r_S}{K_S} S^2 - \beta(a + F_n) S e^{-d(t-t_n^+)}. \end{aligned}$$

Hence differentiating with respect to t gives

$$\frac{d^2S}{dt^2} = r_S \frac{dS}{dt} - 2\frac{r_S}{K_S} S \frac{dS}{dt} - \beta(a + F_n)\frac{dS}{dt} e^{-d(t-t_n^+)} + d\beta(a + F_n) S e^{-d(t-t_n^+)}.$$

At the possible minimal spraying strategy, $\frac{dS}{dt} = 0$, and so

$$\frac{d^2S}{dt^2} = d\beta(a + F_n) S e^{-d(t-t_n^+)} > 0.$$

Hence the minimal spraying strategy is given by (3.8). □

Lemma 3.2.6. *If there exists an minimal spraying time then it is unique.*

Proof. Let

$$f(t) := \left(r_S - \beta(a+F_n)e^{-d(t-t_n^+)}\right)\left(r_S S_n \int_{t_n^+}^{t} \hat{E}(u)\,du + K_S\hat{E}(t_n^+)\right) - r_S S_n \hat{E}(t) \quad (3.10)$$

for $t > t_n^+$. Then the minimal spraying time is given by $f(t) = 0$. Now

$$\begin{aligned} f'(t) &= \left(d\beta(a+F_n)e^{-d(t-t_n^+)}\right)\left(r_S S_n \int_{t_n^+}^{t} \hat{E}(u)\,du + K_S\hat{E}(t_n^+)\right) \\ &\quad + \left(r_S - \beta(a+F_n)e^{-d(t-t_n^+)}\right) r_S S_n \hat{E}(t) - r_S S_n \hat{E}'(t), \end{aligned}$$

but

$$\hat{E}'(t) = \left(r_S - \beta(a+F_n)e^{-d(t-t_n^+)}\right)\hat{E}(t).$$

So finally we have that

$$\begin{aligned} f'(t) &= \left(d\beta(a+F_n)e^{-d(t-t_n^+)}\right)\left(r_S S_n \int_{t_n^+}^{t} \hat{E}(u)\,du + K_S\hat{E}(t_n^+)\right) \\ &> 0 \quad \forall t > t_n^+. \end{aligned}$$

Therefore f is strictly increasing and thus, if there exists a $t > t_n^+$ such that $f(t) = 0$ then it is unique. □

Lemma 3.2.7. *An minimal spraying time exists for $t > t_n^+$ if, and only if,*

1. $\xi < 0$ *if* $S_n < K_S$
2. $\xi < 0$ *and* $\zeta > 0$ *if* $S_n > K_S$

where

$$\begin{aligned} \xi &= r_S(K_S - S_n) - \beta K_S(a+F_n) \\ \zeta &= r_S S_n \beta(F_n + a) \lim_{t\to\infty}\left[\int_{t_n^+}^{t} e^{-d(u-t_n^+)}\hat{E}(u)\,du - e^{d(t-t_n^+)}\int_{t_n^+}^{t}\hat{E}(u)\,du\right] \\ &\quad + r_S\hat{E}(t_n^+)(K_S - S_n). \end{aligned}$$

Proof. Now,

$$\begin{aligned} f(t_n^+) &= (r_S - \beta(a+F_n))K_S\hat{E}(t_n^+) - r_S S_n\hat{E}(t_n^+) \\ &= \hat{E}(t_n^+)\left\{r_S(K_S - S_n) - \beta K_S(a+F_n)\right\}. \end{aligned}$$

Thus, $f(t_n^+) < 0$ if, and only if, $\xi < 0$. Since f is a strictly increasing function (see Lemma 3.2.6) then it is necessary to force $f(t_n^+) < 0$ for a solution to exist. Then we will have necessary and sufficient conditions for the existence of a solution if $\lim_{t\to\infty} f(t) > 0$.

$$\begin{aligned}
\lim_{t\to\infty} f(t) &= \lim_{t\to\infty}\left[\left(r_S-\beta(a+F_n)e^{-d(t-t_n^+)}\right)\left(r_S S_n\int_{t_n^+}^{t}\hat{E}(u)\,du+K_S\hat{E}(t_n^+)\right)\right.\\
&\quad\left.-r_S S_n\hat{E}(t)\right]\\
&= \lim_{t\to\infty}\left[r_S S_n\left(\int_{t_n^+}^{t} r_S\hat{E}(u)\,du-\hat{E}(t)\right)\right]\\
&\quad-\beta(F_n+a)\lim_{t\to\infty}\left[e^{-d(t-t_n^+)}r_S S_n\int_{t_n^+}^{t}\hat{E}(u)\,du\right]\\
&\quad-\lim_{t\to\infty}\left[\beta(F_n+a)e^{-d(t-t_n^+)}K_S\hat{E}(t_n^+)\right]+r_S K_S\hat{E}(t_n^+)\\
&= r_S S_n\lim_{t\to\infty}\left[\int_{t_n^+}^{t}\left(r_S\hat{E}(u)-\hat{E}'(u)\right)du\right]-r_S\hat{E}(t_n^+)(S_n-K_S)\\
&\quad-\lim_{t\to\infty}\left[\beta(F_n+a)e^{-d(t-t_n^+)}r_S S_n\int_{t_n^+}^{t}\hat{E}(u)\,du\right].
\end{aligned}$$

Thus

$$\lim_{t\to\infty} f(t)=\zeta.$$

But,

$$e^{-d(t-t_n^+)}\le e^{-d(u-t_n^+)}\text{ for all }u\in[t_n^+,t],$$

and hence

$$\int_{t_n^+}^{t} e^{-d(u-t_n^+)}\hat{E}(u)\,du-e^{-d(t-t_n^+)}\int_{t_n^+}^{t}\hat{E}(u)\,du>0.$$

Thus if

1. $S_n<K_S$ then $\zeta>0$ always,
2. $S_n>K_S$ then we must impose that $\zeta>0$.

Hence the result follows. □

The analytical result presented in Lemma 3.2.7 is demonstrated numerically in Figure 3-3. In the model formulation, the parameter values remain constant, and only the spraying times change. Hence, each spraying time corresponds to an initial condition ($S(t_n^+)=S_n$ and $F(t_n^+)=F_n+a$) in the phase plane. Therefore, depending on which region the initial condition lies determines the quantitative behaviour of the system.

It is trivial to show that the nullcline drawn in the phase plane corresponds to $\xi = 0$, and that the minimal spraying time is given by the ∞-cline (i.e. where the solution trajectory (phase curve) crosses the nullcline).

Hence, $\xi > 0$ corresponds to the region under the nullcline, and as indicated from Figure 3-3 the solution trajectory does not cross the ∞-cline and so there is no minimal spraying time.

In the region above the nullcline ($\xi < 0$) and initial condition, $S_n < K_S$ the solution trajectory must cross the nullcline in order to reach the stable steady state. Therefore, in this region an minimal spraying time must exist.

If the initial condition, $S_n > K_S$, then the solution trajectory may, or may not, cross the nullcline, and therefore there may, or may not, be an minimal spraying strategy. Hence there must be a separatrix which marks a boundary between the two cases. In Lemma 3.2.7 I proved that if $S_n > K_S$ then the existence of the minimal spraying strategy depended on the sign of ζ. Therefore, I conjecture that the separatrix is given by $\zeta = 0$; however, I am unable to prove this to be true or false.

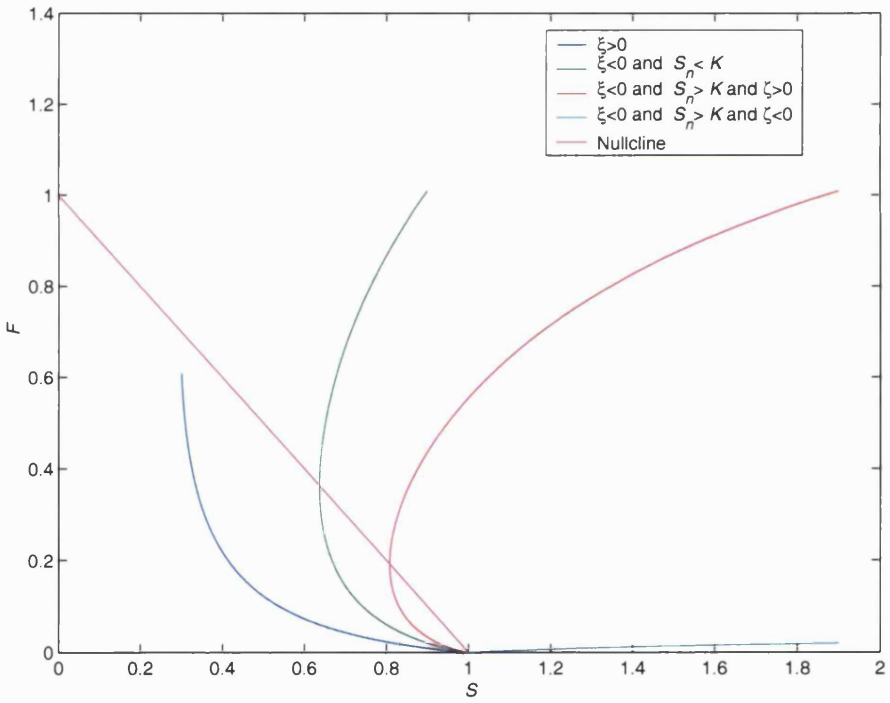


Figure 3-3: Phase Plane of Model 3.2. The parameter values are $r_S = K_S = \beta = d = 1$.

With Resistance

When resistance is included in the model, then (3.1) becomes

$$\frac{dS}{dt} = r_S S\left(1 - \frac{S + \alpha_1 R}{K_S}\right) - \beta SF \tag{3.11a}$$

$$\frac{dR}{dt} = r_R R\left(1 - \frac{R + \alpha_2 S}{K_R}\right) \tag{3.11b}$$

$$\frac{dF}{dt} = -dF \tag{3.11c}$$

with the conditions

$$S(t_n^-) = S(t_n^+) = S_n \tag{3.11d}$$

$$R(t_n^-) = R(t_n^+) = R_n \tag{3.11e}$$

$$F(t_n^+) = F(t_n^-) + a \tag{3.11f}$$

$$F(t_n^-) = F_n. \tag{3.11g}$$

Equation (3.11) cannot be solved explicitly. Therefore, the analysis carried out in the No Resistance section cannot be conducted here. Hence, I resort to detailed numerical simulations to demonstrate the types of behaviour that (3.11) exhibits.
To simplify the simulations, I rescale the state variables and model parameters using the following substitutions

$$S = K_S x \quad R = K_R y \quad \tau = r_S t \quad z = \frac{\beta}{r_S} F$$
$$\theta_1 = \frac{\alpha_1 K_R}{K_S} \quad \theta_2 = \frac{\alpha_2 K_S}{K_R} \quad r = \frac{r_R}{r_S} \quad b = \frac{d}{r_S}$$

to get the model

$$\frac{dx}{d\tau} = x(1 - x - \theta_1 y) - xz \tag{3.12a}$$

$$\frac{dy}{d\tau} = ry(1 - y - \theta_2 x) \tag{3.12b}$$

$$\frac{dz}{d\tau} = -bz. \tag{3.12c}$$

Since the chemical insecticide level always decays to zero with a single application, the resulting system tends to the Lotka-Volterra competition model as $t \to \infty$. Hence, using a single application Model 3.12 has four steady states:

$(x^*, y^*, z^*) = (0,0,0)$ which is never stable

$(x^*, y^*, z^*) = (1,0,0)$ which is stable if, and only if, $\theta_2 > 1$

$(x^*, y^*, z^*) = (0,1,0)$ which is stable if, and only if, $\theta_1 > 1$

$(x^*, y^*, z^*) = \left(\frac{1-\theta_1}{1-\theta_1\theta_2}, \frac{1-\theta_2}{1-\theta_1\theta_2}, 0\right)$ which is stable if, and only if, $\theta_1 < 1$ and $\theta_2 < 1$.

There are four parameter regions that exhibit different long-term behaviours, and therefore, there are four regions to investigate the long-term behaviours for the periodic spraying strategy, $t_n = nh$ for $h > 0$.

$\theta_1 < 1, \theta_2 < 1$: In this case under a single application of insecticide, the non-trivial steady state is globally stable and

$$\frac{1-\theta_1}{1-\theta_1\theta_2} < 1 \quad \text{and} \quad \frac{1-\theta_2}{1-\theta_1\theta_2} < 1.$$

Moreover,

$$\frac{1-\theta_1}{1-\theta_1\theta_2} + \frac{1-\theta_2}{1-\theta_1\theta_2} > 1.$$

Using a periodic spraying strategy causes an increase in the resistants and a decrease in the susceptibles. Decreasing the time between sprays (decreasing h) causes a greater increase in the resistants and a greater decrease in the susceptibles. If the time between sprays is sufficiently small then the susceptibles can be eradicated, and the resistants go to carrying capacity, as indicated in Figure 3-4. This effect is due to the fact that both susceptibles and resistants are weak competitors, and the additional insecticide causes a decrease in the susceptibles growth rate, and hence the relative competitive ability of the resistants increases, thus causing the resistants to out-compete the susceptibles.

$\theta_1 < 1, \theta_2 > 1$: In this case the susceptibles are strong competitors and the resistants are weak competitors. Hence, in the absence of a pulsed spraying strategy, the susceptibles out-compete the resistants. So how does the insecticide affect this relationship? Is it possible to eradicate the aphid population?

From the extensive numerical experiments that I have carried out, I have found no indication that the aphid population can be completely eradicated. However, as indicated in Figure 3-5 (a) it is possible to reduce the total aphid numbers. We see that if the time between sprays is sufficiently long then the susceptibles are still able to out-compete the resistants but suffer losses due to the presence of the insecticide. Hence the susceptibles are unable to approach carrying capacity. On the other hand, if the time between sprays is sufficiently short then the susceptibles lose their competitive edge and the resistants out-compete the susceptibles. Moreover, it doesn't seem possible to drive the susceptibles arbitrarily close to eradication by decreasing the times between sprays before the competitive switch takes place. Thus from a control point of view, the times between sprays may be crucial in devising a control strategy for the pest insects.

In Figure 3-5 (b) we see that different initial conditions affects which biotype

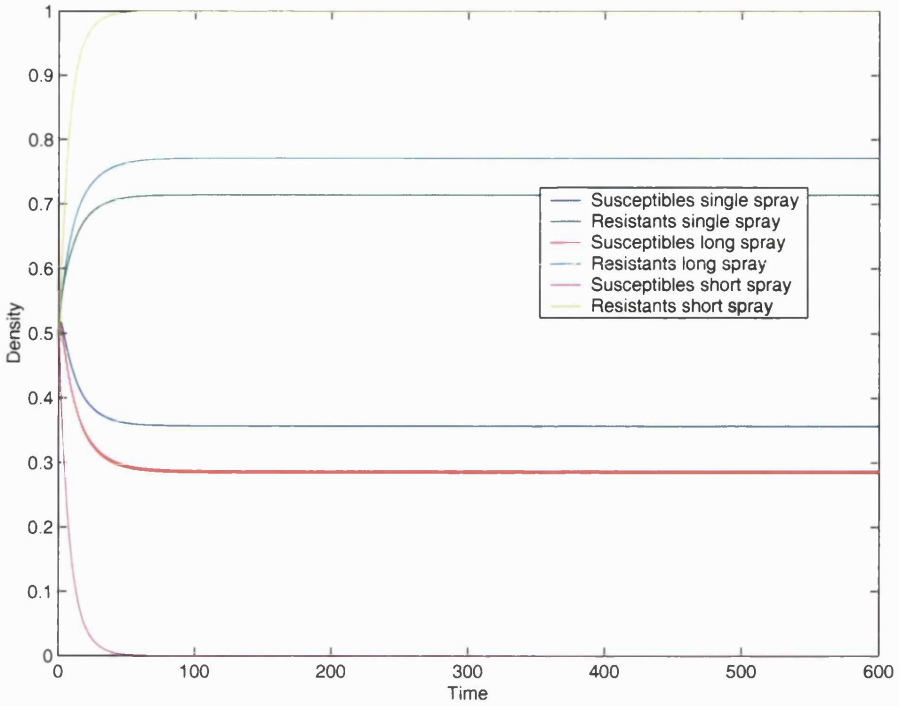


Figure 3-4: $\theta_1 < 1, \theta_2 < 1$. The parameter values are $\theta_1 = 0.9, \theta_2 = 0.8, b = 10, r = 1, a = 0.2$. In the case where the time between sprays in long $h = 1$, and the case where the time between sprays is short $h = 0.1$.

out-competes the other. This is not the case in the single application strategy, where the susceptibles only steady state is globally stable.

$\theta_1 > 1, \theta_2 < 1$: In this case the susceptibles are weak competitors and the resistants are strong competitors. Thus using a single application of the insecticide, the resistants will always out-compete the susceptibles. Hence introducing the pulsed insecticide will have no effect on the long-term outcome of the model. However, with the introduction of the pulsed insecticide, the susceptibles will be eradicated more quickly, and the resistants approach carrying capacity at a faster rate. This is due to the inhibiting effect on the susceptibles growth rate.

$\theta_1 > 1, \theta_2 > 1$: In the case where no insecticide is applied, this system reverts to the Lotka-Volterra competition model, where both species are strong competitors. Hence the system is bistable, and so the steady state that is approached depends on the initial populations as shown in Figure 3-6. One can clearly see the separatrix that exists (the proof that such a separatrix exists can be found in Langa, Robinson & Suárez (2003)) passes through the nullclines intersection. Although this separatrix can be shown to exist, there is no explicit equation for the separatrix.

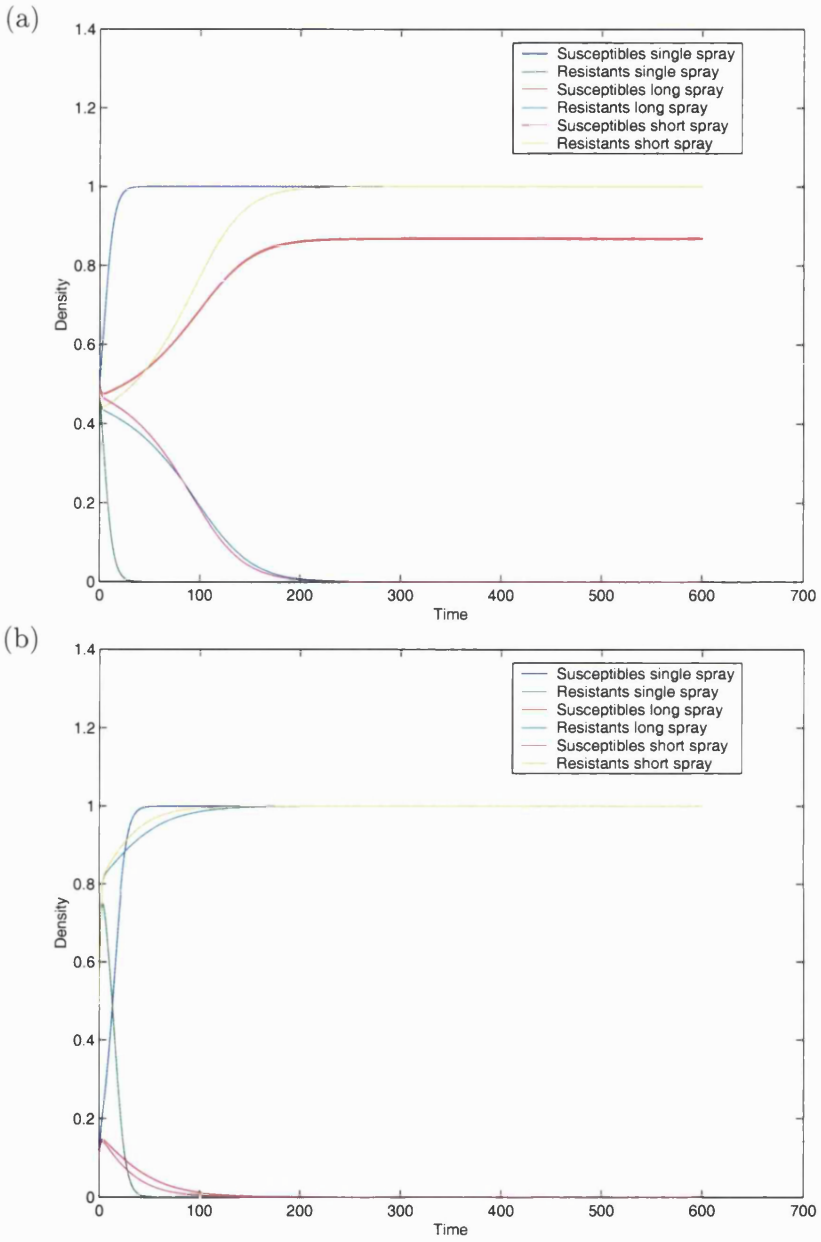


Figure 3-5: $\theta_1 < 1, \theta_2 > 1$. The parameter values are $\theta_1 = 0.9, \theta_2 = 1.2, b = 10, r = 1, a = 0.2$. In the case where the time between sprays in long $h = 0.15$, and the case where the time between sprays is short $h = 0.14$. In both plots, the parameter values are the same, only the initial population densities differ.

By studying the phase planes, it is simple to show that a single release of chemical insecticide has no effect on the long-term behaviour of the system, and the separatrix does not change in the (x, y) plane. However, introducing a periodically pulsed input of insecticide can have a significant effect on the long-term behaviour.

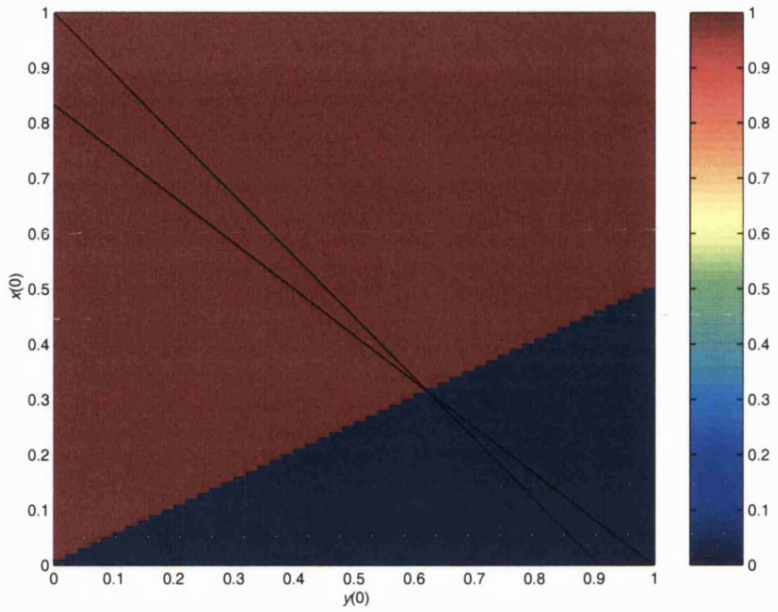


Figure 3-6: $\theta_1 > 1, \theta_2 > 1$. The parameter values are $\theta_1 = 1.1, \theta_2 = 1.2, b = 10, r = 1$. The two black lines are the non-zero nullclines for the system. The colouring indicates the steady state value approached by the susceptibles from the specified initial populations. Red indicates that the susceptibles have gone to a positive steady state, whereas blue denotes that the susceptibles have died out. The plot for the resistants is the negative of the plot for the susceptibles, since both species are strong competitors.

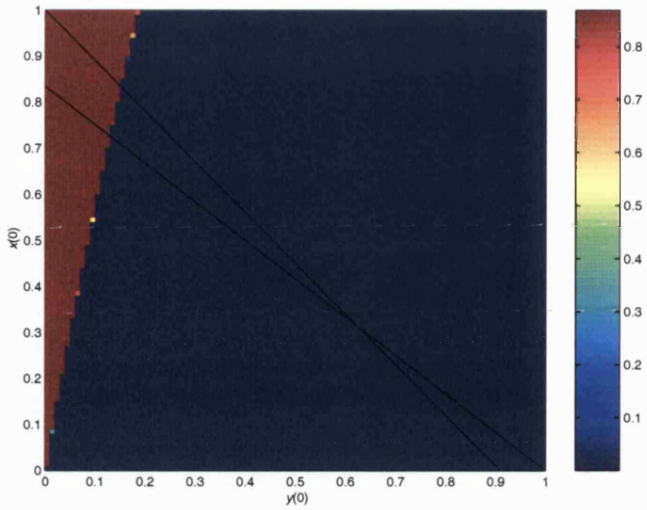
In Figure 3-7 we immediately see that the basins of attraction have changed when using a pulsed insecticide. As the time between pulses decreases, the basin of attraction for the susceptibles to go to a positive steady state decreases. For the susceptibles to out-compete the resistants, the initial population of susceptibles must be significantly larger than that of the resistants. Intuitively, we see that as the time between sprays decreases, the positive steady state levels for the susceptibles also decreases.

3.2.3 Fungal Pathogen

The main difference between the fungal pathogen and the chemical insecticide (from a modelling point of view) is that secondary infections may occur, thus we assume that $\omega > 0$ in equations (3.1).

Let us first consider the case where no resistance develops.

(a)



(b)

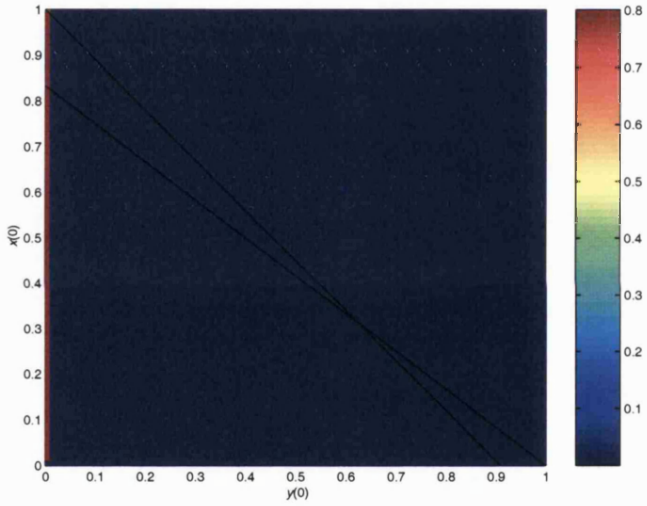


Figure 3-7: $\theta_1 > 1, \theta_2 > 1$. The parameter values are $\theta_1 = 1.1, \theta_2 = 1.2, b = 10, r = 1, a = 0.2$. In (a) the time between sprays is relatively long, $h = 0.15$, and in (b) the time between sprays is relatively short, $h = 0.1$. The colour coding is the same as in Figure 3-6.

No Resistance

With $R(t) \equiv 0$ and $\omega > 0$, Model 3.1 becomes

$$\frac{dS}{dt} = r_S S\left(1 - \frac{S}{K_S}\right) - \beta SF \tag{3.13a}$$

$$\frac{dF}{dt} = -dF + \omega\beta SF \tag{3.13b}$$

for $t \in (t_n^+, t_{n+1}^-)$, with

$$S(t_n^-) = S(t_n^+) = S_n \tag{3.13c}$$

$$F(t_n^+) = a + F(t_n^-) = a + F_n. \tag{3.13d}$$

Since $\omega > 0$ the system is coupled, and deriving an explicit stroboscopic map is impossible. Therefore, I use Floquet theory (as used in Sherratt (1995)) to derive conditions on the stability of periodic solutions.
Let us consider a system of equations

$$\frac{dS}{dt} = f(S, F, t) \tag{3.14a}$$

$$\frac{dF}{dt} = g(S, F, t) \tag{3.14b}$$

where the parameters in f and g vary in a 'square-tooth' manner, that is, f and g have different dynamics on $nT < t < (n + 1/2)T$ and $(n + 1/2)T < t < (n + 1)T$ $(n \epsilon \mathbb{N})$. Here T is the period. This 'square-toothed' manner corresponds to the fungus being switched on and off. Specifically I will take

$$f(S, F, t) = f_1(S, F) = r_S S\left(1 - \frac{S}{K_S}\right) - \beta SF \tag{3.15a}$$

$$g(S, F, t) = g_1(S, F) = \omega\beta SF - dF + a \tag{3.15b}$$

on $nT < t < (n + 1/2)T$ as the on-state and

$$f(S, F, t) = f_2(S, F) = r_S S\left(1 - \frac{S}{K_S}\right) - \beta SF \tag{3.15c}$$

$$g(S, F, t) = g_2(S, F) = \omega\beta SF - dF \tag{3.15d}$$

on $(n + 1/2)T < t < (n + 1)T$ as the off-state. I assume that there exists a periodic solution $(S, F) = (S_s(t), F_s(t))$ with the same period T. Ecologically this makes sense since it is this pulsing of the fungal pathogen that drives the long-term dynamics. Moreover, I have extensive numerical evidence that suggests at such a solution. The idea now is look for stability of such periodic solutions as in Sherratt (1995). To do this I look at two extreme cases. The first is where the period is very small which corresponds to frequent application of the fungal pathogen. The second is where the period is very long.

$T \to 0$: Let's consider what happens as the period tends to zero. Then as derived by Sherratt (1995), we have stability if, and only if,

$$\bar{a} + \bar{d} < 0 \text{ and } \bar{a}\bar{d} - \bar{b}\bar{c} > 0 \tag{3.16}$$

where

$$\begin{aligned}
\bar{a} &= 1/2\left(\left.\frac{\partial f_1}{\partial S}\right|_{(S_0,F_0)} + \left.\frac{\partial f_2}{\partial S}\right|_{(S_0,F_0)}\right)\\
\bar{b} &= 1/2\left(\left.\frac{\partial f_1}{\partial F}\right|_{(S_0,F_0)} + \left.\frac{\partial f_2}{\partial F}\right|_{(S_0,F_0)}\right)\\
\bar{c} &= 1/2\left(\left.\frac{\partial g_1}{\partial S}\right|_{(S_0,F_0)} + \left.\frac{\partial g_2}{\partial S}\right|_{(S_0,F_0)}\right)\\
\bar{d} &= 1/2\left(\left.\frac{\partial g_1}{\partial F}\right|_{(S_0,F_0)} + \left.\frac{\partial g_2}{\partial F}\right|_{(S_0,F_0)}\right)
\end{aligned}$$

and (S_0, F_0) satisfies

$$f_1(S_0, F_0) + f_2(S_0, F_0) = g_1(S_0, F_0) + g_2(S_0, F_0) = 0. \tag{3.17}$$

Now, the solutions of (3.17) are

$$(S_0, F_0) = \left(0, \frac{a}{2d}\right), (S_0^*, F_0^*) \tag{3.18}$$

where S_0^* satisfies the quadratic

$$S_0^{*2} - \left(\frac{1}{K_S} + \frac{d}{\beta\omega}\right) S_0^* + \frac{K_S}{2\omega r_S}\left(\frac{2dr_S}{\beta} - a\right) = 0$$

and F_0^* is given by

$$F_0^* = \frac{r_S}{\beta}\left(1 - \frac{S_0^*}{K_S}\right).$$

Notice that in order for the steady state $(S_0, F_0) = (S_0^*, F_0^*)$ to be biologically realistic we must impose that

$$r_S - \frac{\beta a}{2d} > 0, \tag{3.19}$$

by Figure 3-8. Note that if (3.19) holds then $S_0^* < K_S$ and so $F_0^* > 0$. Now we have that

$$\begin{aligned}
\bar{a} &= r_S\left(1 - \frac{2S_0}{K_S}\right) - \beta F_0\\
\bar{b} &= -\beta S_0\\
\bar{c} &= \omega\beta F_0\\
\bar{d} &= \omega\beta S_0 - d.
\end{aligned}$$

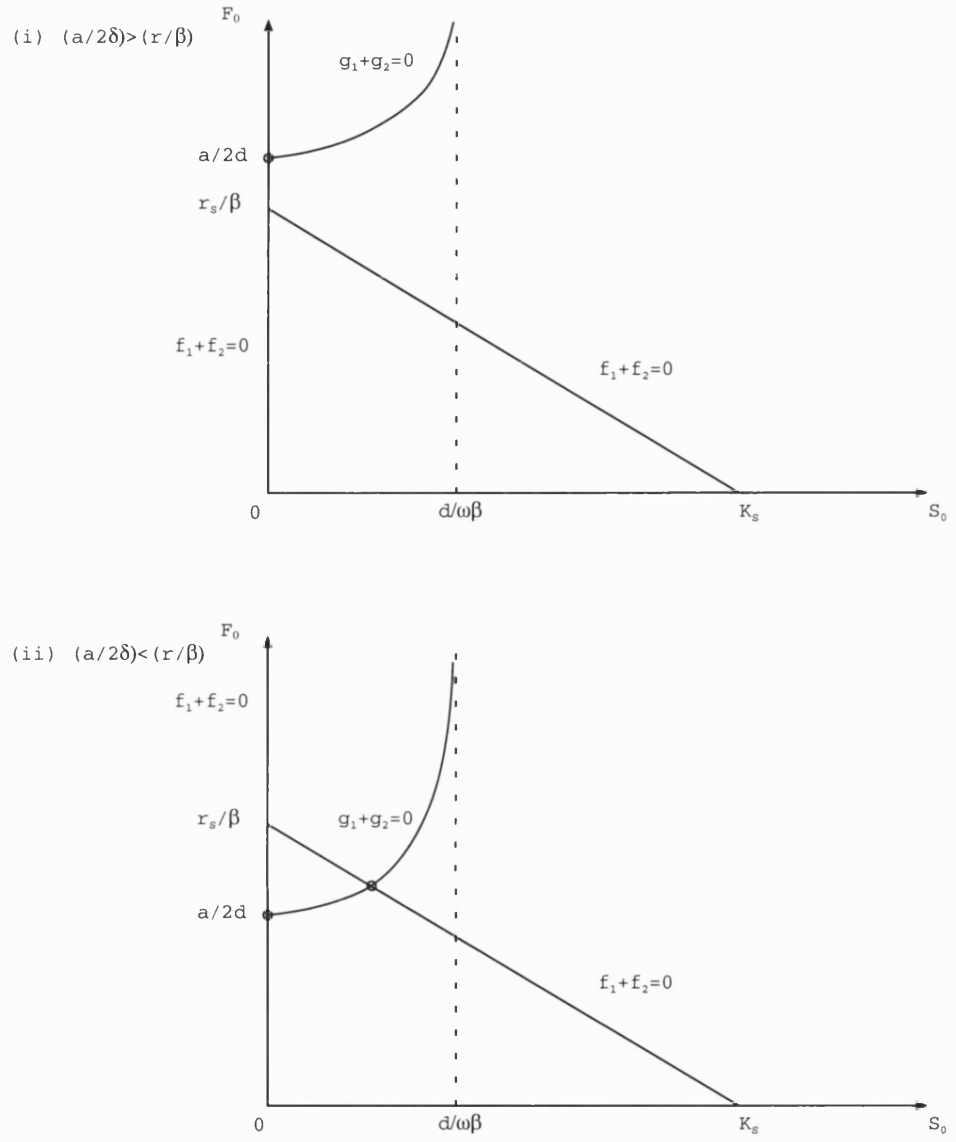


Figure 3-8: Solutions to the steady state of the sum of the kinetic terms (3.17).

So for $(S_0, F_0) = (0, a/2d)$ the stability conditions (3.16) give

$$\left(r_S - \frac{\beta a}{2d}\right) - d < 0 \quad \text{and} \quad -d\left(r_S - \frac{\beta a}{2d}\right) > 0.$$

Hence the periodic solution is stable if, and only if,

$$r_S - \frac{\beta a}{2d} < 0 \tag{3.20}$$

So now let's consider the other steady state solution $(S_0, F_0) = (S_0^*, F_0^*)$. Then the stability conditions (3.16) become

$$-\frac{r_S}{K_S}S_0^* + (\omega\beta S_0^* - d) < 0 \quad \text{and} \quad -\frac{r_S}{K_S}S_0^*(\omega\beta S_0^* - d) + \omega\beta^2 S_0^* F_0^* > 0.$$

Now, from Figure 3-8 we see that $S_0^* < \frac{d}{\omega\beta}$, which is equivalent to $\omega\beta S_0^* - d < 0$. Thus if the solution is biologically relevant, that is,

$$r_S - \frac{\beta a}{2d} > 0 \tag{3.21}$$

then the periodic solution will be stable.

T $\to \infty$: Let us now consider what happens when the period becomes very long. To analyse this periodic behaviour we must impose two additional conditions on the kinetics, for $i = 1, 2$, $f_i(S, F)$ and $g_i(S, F)$ must have a unique steady state in the first positive quadrant that is globally attracting and linearly stable. Under this assumption, in the limit as $T \to \infty$, any periodic solution will tend towards $(S_{s,1}, \Phi_{s,1})$ and $(S_{s,2}, \Phi_{s,2})$ in the first and second half of each period respectively.
Now define

$$a_i = \left.\frac{\partial f_i}{\partial S}\right|_{(S_{s,i}, F_{s,i})} \tag{3.22a}$$

$$b_i = \left.\frac{\partial f_i}{\partial F}\right|_{(S_{s,i}, F_{s,i})} \tag{3.22b}$$

$$c_i = \left.\frac{\partial g_i}{\partial S}\right|_{(S_{s,i}, F_{s,i})} \tag{3.22c}$$

$$d_i = \left.\frac{\partial g_i}{\partial F}\right|_{(S_{s,i}, F_{s,i})} \tag{3.22d}$$

for $i = 1, 2$. Then by Sherratt (1995), $(S_s(t), F_s(t))$ is stable to perturbations if

$$\tau_2(2\tau_1 + \tau_2) + 4\Delta_1 > 0 \tag{3.23a}$$

$$\tau_1(2\tau_2 + \tau_1) + 4\Delta_2 > 0 \tag{3.23b}$$

$$(\tau_2\Delta_1 + \tau_1\Delta_2)(\tau_1 + \tau_2) + (\Delta_1 - \Delta_2)^2 > 0 \tag{3.23c}$$

$$\tau_1 + \tau_2 < 0 \tag{3.23d}$$

where $\tau_i = a_i + d_i$ and $\Delta_i = a_i d_i - b_i c_i$ for $i = 1, 2$.
Let's now consider the on-state. The steady states are given by

$$f_1(S_{s,1}, F_{s,1}) = g_1(S_{s,1}, F_{s,1}) = 0.$$

Thus,

$$(S_{s,1}, F_{s,1}) = \left(0, \frac{a}{d}\right), (S^*_{s,1}, F^*_{s,1})$$

where $(S^*_{s,1}, F^*_{s,1})$ satisfy

$$S^{*\,2}_{s,1} - \left(\frac{1}{K_S} + \frac{d}{\omega\beta}\right) S^*_{s,1} + \frac{K_S}{\omega r_S}\left(\frac{dr_S}{\beta} - a\right) = 0 \quad \text{and} \quad F^*_{s,1} = \frac{r_S}{\beta}\left(1 - \frac{S^*_{s,1}}{K_S}\right).$$

By analysing the Jacobian and the phase plane (see Figure 3-9), it can be shown that $(S_{s,1}, F_{s,1}) = (S^*_{s,1}, F^*_{s,1})$ is biologically realistic and therefore stable if, and only if,

$$r_S - \frac{\beta a}{d} > 0, \tag{3.24}$$

otherwise $(S_{s,1}, F_{s,1}) = \left(0, \frac{a}{d}\right)$ is stable. So, to use the Floquet Theory we must assume (3.24) to be true (otherwise the susceptibles die out).
Now let's consider the off-state. Again, we look for equilibria by setting

$$f_2(S_{s,2}, F_{s,2}) = g_2(S_{s,2}, F_{s,2}) = 0.$$

So the steady states are

$$(S_{s,2}, \Phi_{s,2}) = (0,0), (K_S, 0), \left(\frac{d}{\omega\beta}, \frac{r_S}{\beta}\left[1 - \frac{d}{\omega\beta K_S}\right]\right). \tag{3.25}$$

Notice that in order for the non-trivial steady state to be biologically relevant, we must impose that

$$\omega\beta K_S - d > 0. \tag{3.26}$$

In fact, (3.26) is enough to guarantee stability which can be seen by considering Figure 3-10 and determining the eigenvalues of the Jacobian. Thus to use the Floquet theory, we can impose (3.24) and (3.26) and we have necessary and sufficient conditions for the existence of the equilibrium to lie in the first positive quadrant that are globally and linearly stable.
Now for the periodic solution to be stable we must check conditions (3.23) hold. Recall that τ_i and Δ_i $(i = 1, 2)$ are simply the trace and determinant of the Jacobian respectively. Thus, by the assumption that (3.24) and (3.26) are true we ensure that $\tau_i < 0$ and $\Delta_i > 0$ $(i = 1, 2)$. Hence conditions (3.23) hold. This makes intuitive sense since the period is sufficiently long so that in each half of the period you are tending to a steady state and then switching to another. Thus stability conditions for each equilibria should ensure stability of the periodic solution.

Numerical Simulations: In this brief section I present a few numerical simulations to show some of the typical behaviours that are observed.

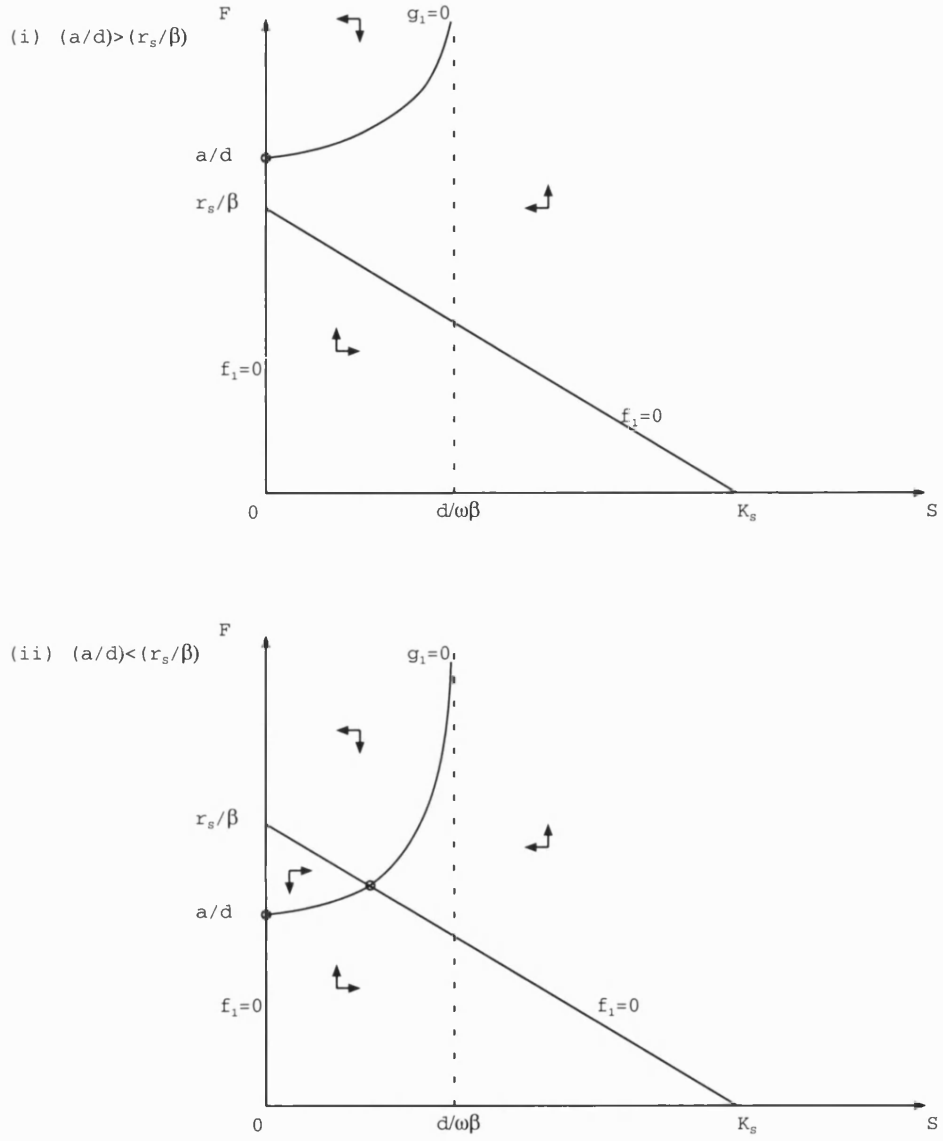


Figure 3-9: The phase planes for the on-state.

In Figure 3-11 (a) and (b) the model parameters remain constant in each plot, but the period, T, changes. In (a) when the period is large we clearly see the two periods. In the first half of the period (the on-state), the parameters have been chosen so that the susceptible aphids tend to zero. However, in the second half of the period (the off-state), the parameters have been chosen so that the susceptible aphids tend to a positive equilibrium with decaying oscillations as one would expect from the phase plane (Figure 3-10). Hence when the fungus is switched off, one would expect to see large fluctuating populations which are not desirable for control.

In (b) where the period is of intermediate length, the susceptible aphids no longer

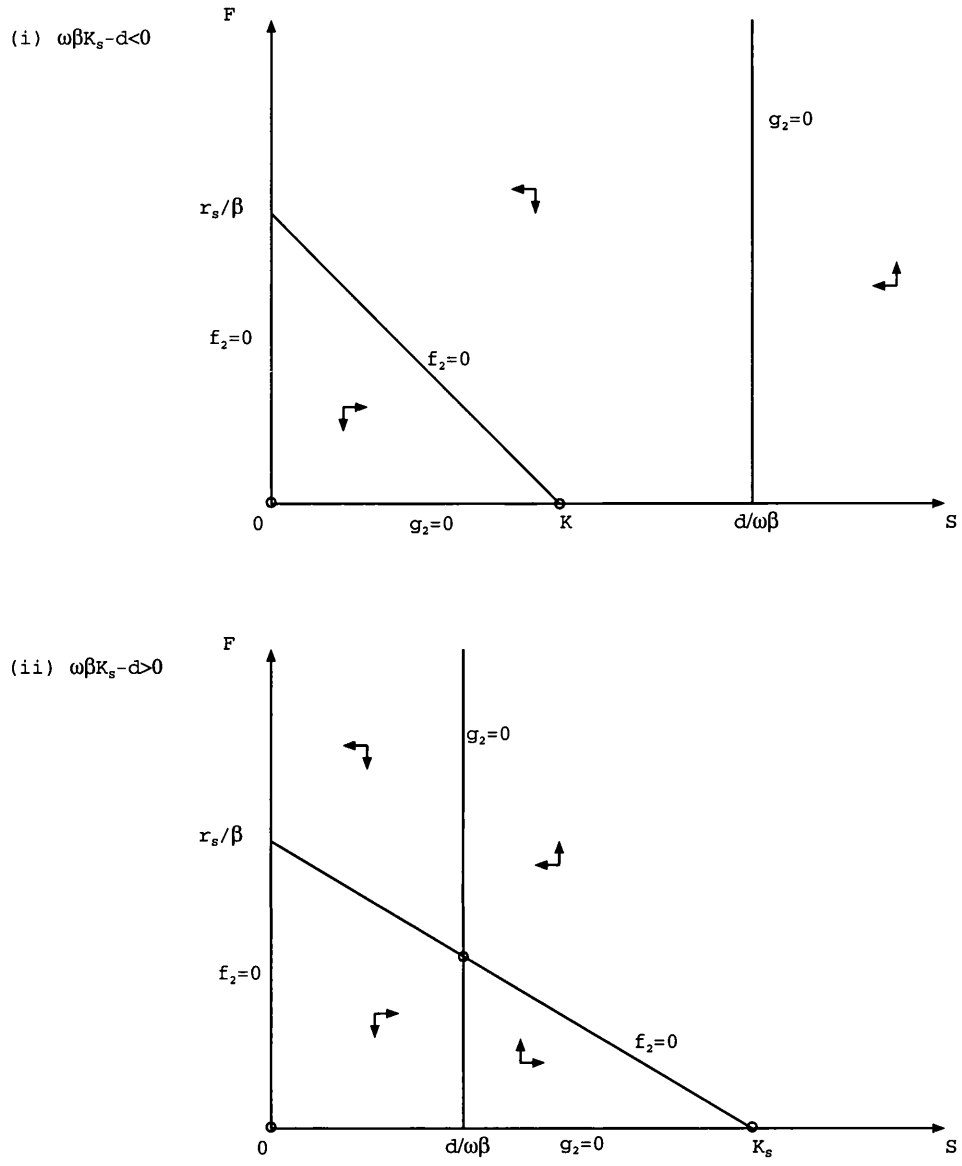


Figure 3-10: The phase planes for the off-state.

approach zero in the on-state since the period is not long enough. However, we do see a stable periodic solution.

In Figure 3-12 the period has been made very small. In (a) the parameters have been chosen so that (3.21) holds. Then as the theory predicts, as $T \to 0$ there will be a stable positive periodic solution (see inlay). Notice that in both Figure 3-11 (b) and Figure 3-12 (a) the model parameters remain the same, and only the period differs.

In Figure 3-12 (b) the intrinsic growth rate of the susceptible aphids has decreased so that condition (3.21) is violated, and we see that the aphids die out, and the solution is not periodic.

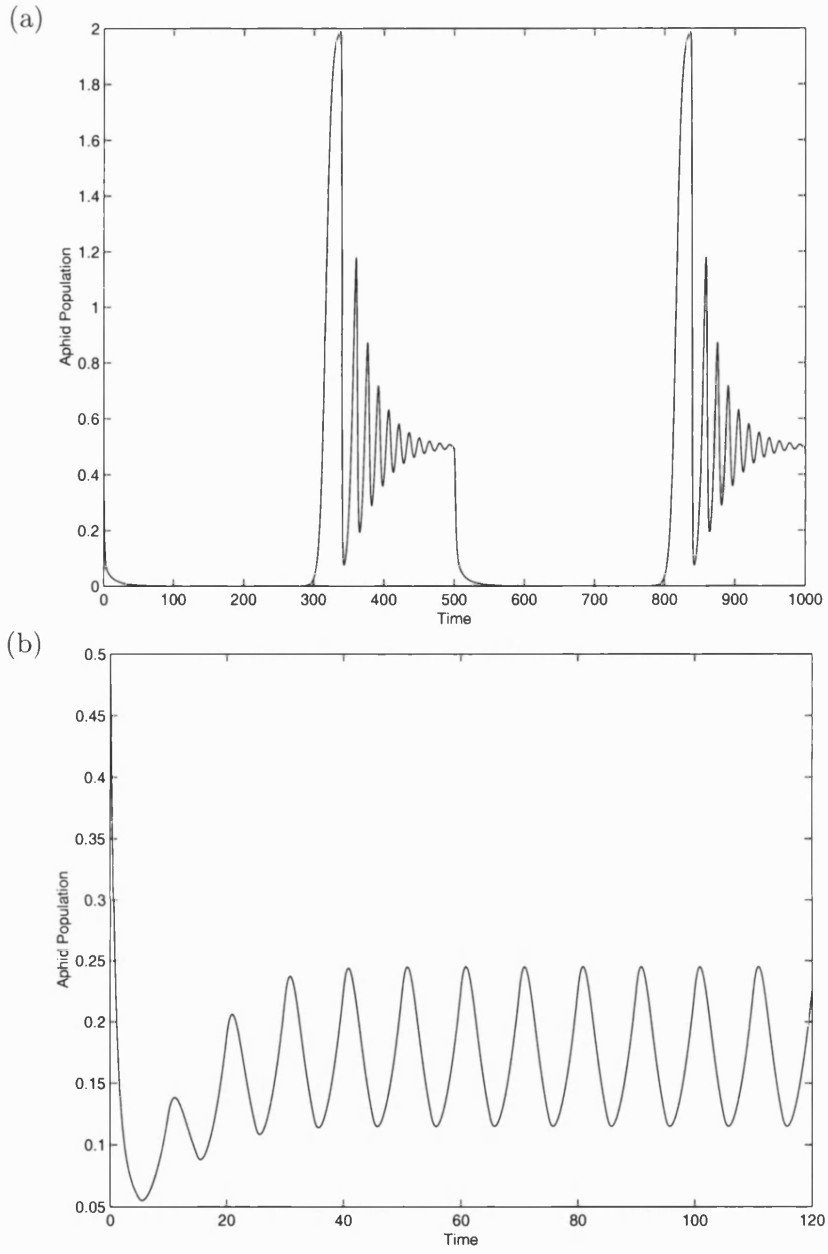


Figure 3-11: Two numerical simulations of Model 3.14 where f and g are given by (3.15). Only the period, T, differs in each plot; in (a) $T = 500$ and in (b) $T = 10$. The model parameters for each plot are $r_S = 0.25$, $K_S = 2$, $\beta = 1$, $d = 1$, $a = 0.3$ and $\omega = 2$.

Clearly the long-term behaviour of the pulsed system depends on the fungus dynamics. It is clear that if the pathogen is sufficiently virulent (i.e. β is sufficiently large) such that

$$r_S - \frac{\beta a}{2d} < 0$$

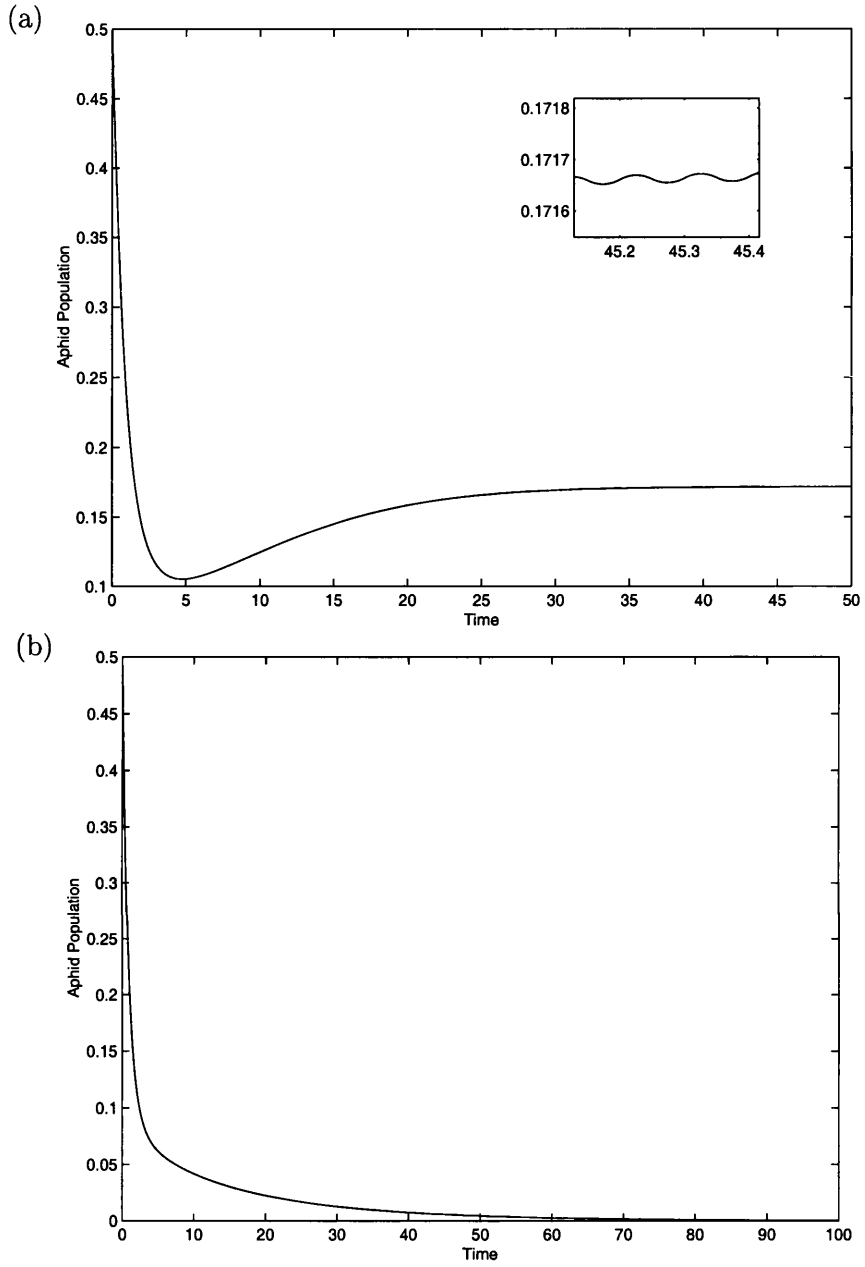


Figure 3-12: Two numerical simulations of Model 3.14 where f and g are given by (3.15). Only the intrinsic growth rate differs in each plot, and the period is $T = 0.1$ for each plot. The model parameters for each plot are $K_S = 2$, $\beta = 1$, $d = 1$, $a = 0.3$, $\omega = 2$; and in (a) $r_S = 0.25$, and in (b) $r_S = 0.1$.

then the pathogen will always be able to eradicate the susceptible hosts independent of the period.

With Resistance

In the following section I consider a fungal pathogen where resistant insects are present. So that analogies can be made with the previous section, I consider the model:

$$\frac{dS}{dt} = f(S,R,F,t) \tag{3.27a}$$
$$\frac{dR}{dt} = g(S,R,F,t) \tag{3.27b}$$
$$\frac{dF}{dt} = h(S,R,F,t) \tag{3.27c}$$

where

$$f(S,R,F,t) = f_1(S,R,F) = r_S S\left(1-\frac{S+\alpha_1 R}{K_S}\right) - \beta SF \tag{3.28a}$$
$$g(S,R,F,t) = g_1(S,R,F) = r_R R\left(1-\frac{R+\alpha_2 S}{K_R}\right) \tag{3.28b}$$
$$h(S,R,F,t) = h_1(S,R,F) = \omega\beta SF - dF + a \tag{3.28c}$$

on $nT < t < (n+1/2)T$ as the on-state and

$$f(S,R,F,t) = f_2(S,R,F) = r_S S\left(1-\frac{S+\alpha_1 R}{K_S}\right) - \beta SF \tag{3.28d}$$
$$g(S,R,F,t) = g_2(S,R,F) = r_R R\left(1-\frac{R+\alpha_2 S}{K_R}\right) \tag{3.28e}$$
$$h(S,R,F,t) = h_2(S,R,F) = \omega\beta SF - dF \tag{3.28f}$$

on $(n+1/2)T < t < (n+1)T$ as the off-state. Due to the complexity of this system, I will only show some typical numerical solutions. To reduce the number of parameters, I rescale (3.28) using the following substitutions

$$S = K_S x \quad R = K_R y \quad \tau = r_S t \quad z = \frac{\beta}{r_S}F \quad \theta_1 = \frac{\alpha_1 K_R}{K_S}$$
$$\theta_2 = \frac{\alpha_2 K_S}{K_R} \quad r = \frac{r_R}{r_S} \quad b = \frac{d}{r_S} \quad c = \frac{\omega\beta K_S}{r_S} \quad A = \frac{a\beta}{r_S^2}$$

to get the model

$$\frac{dx}{d\tau} = x(1-x-\theta_1 y) - xz \tag{3.29a}$$
$$\frac{dy}{d\tau} = ry(1-y-\theta_2 x) \tag{3.29b}$$
$$\frac{dz}{d\tau} = -bz + cxz + A \tag{3.29c}$$

on $nT < t < (n + 1/2)T$ and

$$\frac{dx}{d\tau} = x(1 - x - \theta_1 y) - xz \tag{3.29d}$$

$$\frac{dy}{d\tau} = ry(1 - y - \theta_2 x) \tag{3.29e}$$

$$\frac{dz}{d\tau} = -bz + cxz \tag{3.29f}$$

on $(n + 1/2)T < t < (n + 1)T$.

As with the numerics carried out with the chemical insecticide, I consider four separate cases when the susceptibles and resistants are either strong or weak competitors.

$\theta_1 < 1, \theta_2 < 1$: In this case both species of aphid are weak competitors. Hence if no pathogen is present then both species will go to positive equilibrium levels. When the period is sufficiently long, both species will go to the same equilibrium levels in the off-state as the equilibrium levels when no pathogen is present. In the on-state, when there is an external application of the fungal pathogen, the susceptibles equilibrium level must be reduced, and so therefore the resistants equilibrium level must increase. This can be seen in Figure 3-13 (a). Although one of species may be significantly reduced in numbers in one of the states, the other species will relatively flourish.

When the period is shortened (as in Figure 3-13 (b)) we see that although the solution converges to a periodic solution (see inlay), the amplitude of the oscillation is small. Since the amplitude is small, it can be approximated by the average of the peak and trough values. This in turn can be approximated by the average of the on- and off-state equilibrium values when the period is sufficiently long.

$\theta_1 < 1, \theta_2 > 1$: In this case, the susceptibles are strong competitors and the resistants are weak competitors. Hence, when no pathogen is present the susceptibles will out-compete the resistants. Thus in the off-state when the period is sufficiently long, the susceptibles will out-compete the resistants. However, in the on-state, the long-term behaviour depends on how much fungal pathogen has been externally applied. If a is sufficiently small (and hence A is sufficiently small) then the susceptibles are able to maintain their competitive advantage, and thus out-compete the resistants, otherwise the resistants out-compete the susceptibles, as shown in Figure 3-14 (a). When the period is sufficiently small, as in Figure 3-14 (b) we see that the periodic solution can be approximated by average equilibrium values from the long period on- and off-states.

On the other hand, if a is sufficiently large then the resistants out-compete the susceptibles and hence go to carrying capacity, and the susceptibles eventually die out.

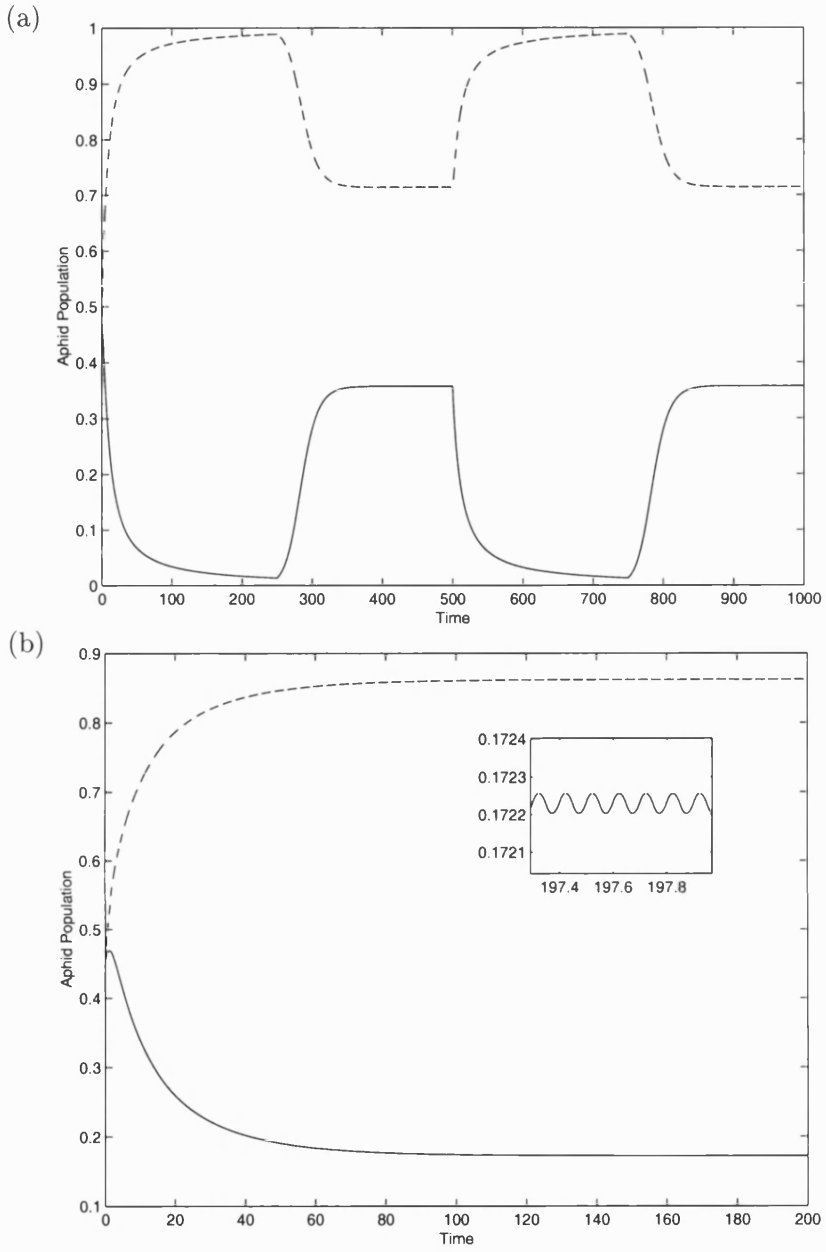


Figure 3-13: Two numerical simulations of Model 3.29. The model parameters for each plot are $r = 1$, $\theta_1 = 0.9$, $\theta_2 = 0.8$, $b = 10$, $c = 2$, $A = 1$; and in (a) $T = 500$, and in (b) $T = 0.1$. The solid line represents susceptibles and the dashed line represents resistants. The inlay in (b) is a magnification of part of the solution. In the on-state, $x^* = 0$ and $y^* = 1$. In the off-state, $x^* \approx 0.35$ and $y^* \approx 0.71$. The average values are $x_0^* \approx 0.175$ and $y_0^* \approx 0.86$.

$\theta_1 > 1, \theta_2 < 1$: In this case the resistants are strong competitors and the susceptibles are weak competitors. Hence in the absence of the pathogen the resistants go to carrying capacity and the susceptibles die out. Thus, the addition of the periodic

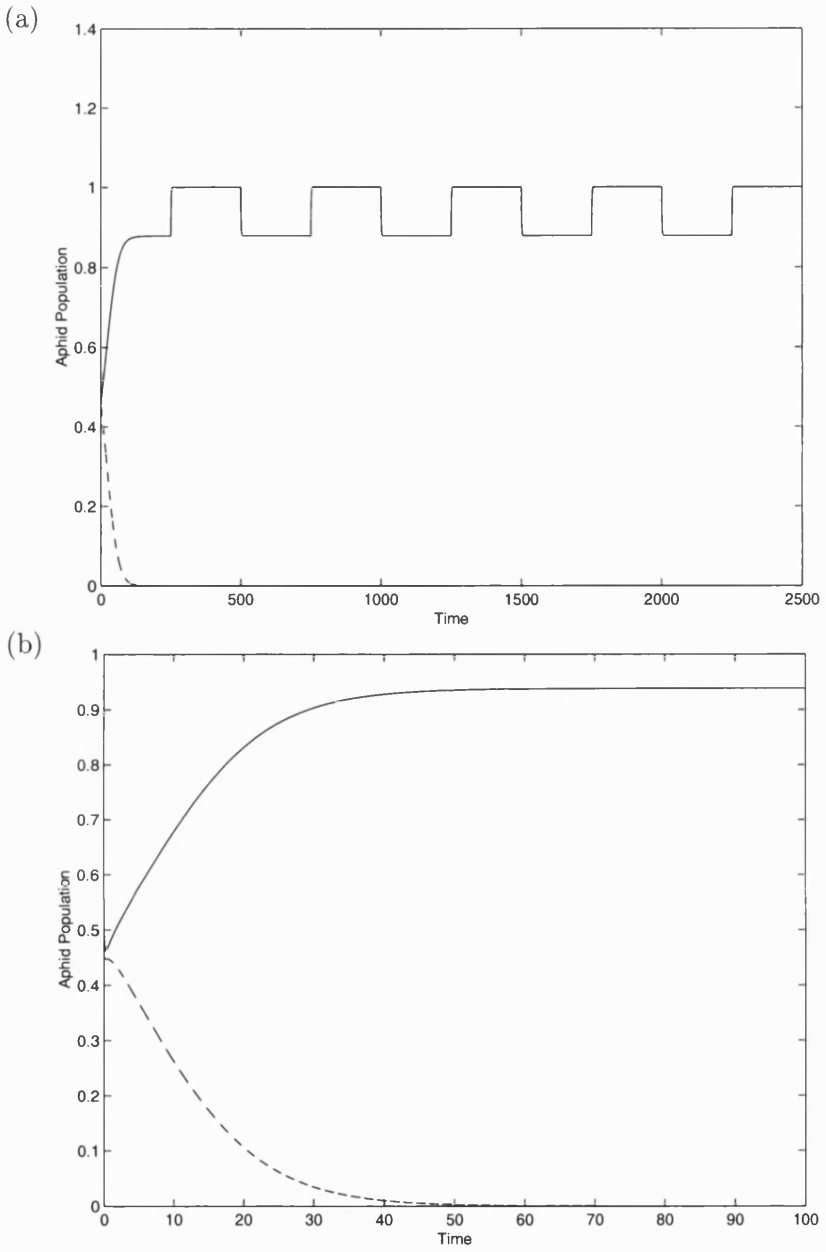


Figure 3-14: Two numerical simulations of Model 3.29. The model parameters for each plot are $r = 1$, $\theta_1 = 0.9$, $\theta_2 = 1.2$, $b = 10$, $c = 2$, $A = 1$; and in (a) $T = 500$, and in (b) $T = 0.1$. The solid line represents susceptibles and the dashed line represents resistants. In the on-state, $x^* \approx 0.80$ and $y^* = 0$. In the off-state, $x^* = 1$ and $y^* = 0$. The average values are $x_0^* = 0.90$ and $y_0^* = 0$.

spraying strategy has no long-term effects on the insect populations.

$\theta_1 > 1, \theta_2 > 1$: In this case both species are strong competitors. In the absence of the pathogen the system is bistable, thus the initial populations of susceptibles and

resistants determines which species out-competes the other. The introduction of the fungal pathogen reduces the basin of attraction for which the susceptibles out-compete the resistants (similar to that seen for the chemical insecticide).

3.3 Conclusions & Discussion

When using a chemical insecticide, the time between sprays is clearly important. If no resistance develops then it is possible to eradicate the pest species providing that the time between sprays is sufficiently short. This critical time may be reduced (so that one doesn't have to spray as often) by increasing the periodic dosage, a, or increasing the coefficient of inoculation, β, or decreasing the decay rate of the chemical insecticide in the environment, d. As the time between sprays increases, the pest population rises but may be maintained at an arbitrarily low level by adjusting the periodic dosage or the time between sprays. The minimal spraying strategy picks a series of times such that the insect pests are eradicated in an efficient manner.

The addition of resistance has a significant effect on the long-term behaviour of the system. If in the absence of the chemical insecticide both pest insect species can coexist, then the periodic spraying strategy can decrease the susceptible population, but will result in an increase in the resistant population. If in the absence of insecticide the susceptibles are able to out-compete the resistants, then applying periodic doses of insecticide will result in lowering the susceptible population as well as allowing the resistant population to out-competed. However, if the time between sprays is too short then the susceptibles will be eradicated, allowing the resistants to go to carrying capacity. If in the absence of the insecticide the resistants are able to out-compete the susceptibles, then the addition of the periodic spraying strategy has little effect on the long-term behaviour.

When using a fungal pathogen, the analysis shows that as the period tends to zero, the susceptible insects can be eradicated if

$$r_S - \frac{\beta a}{2d} < 0 \quad \text{or equivalently} \quad 2 < h^* = \frac{\beta a}{d r_S}. \tag{3.30}$$

This condition is clearly comparable to the condition for eradication when using the chemical insecticide, and therefore eradication can be achieved by applying sufficiently large amounts of the control. Notice that condition (3.30) is independent of ω. This may be due to the fact that as the time between sprays becomes small, the effect of the secondary infections is negligible when compared to the effect of the new applications of pathogen.

However, as the period becomes larger, the secondary infections are required to control the insect population in the off-state (i.e. $\omega\beta K_S - d < 0$) as well as the condition for the on-state (i.e. $1 < h^*$).

Although I have used two different methods for modelling the chemical insecticide and the fungal pathogen, the results can be compared. For example, if the Floquet analysis is carried out for the chemical insecticide model ($\omega = 0$) then as the period tends to zero, the condition for eradication will be the same as for the fungal pathogen (since the result is independent of ω). As the period becomes large, the periodically pulsed chemical insecticide would be unable to eradicate the pests, since in the off-state the chemical would decay allowing the insects to go to carrying capacity. The addition of resistance in the fungal pathogen case leads to similar results as the chemical insecticide case.

The main comparisons between the fungal pathogen and the chemical insecticide are:

- In the absence of resistance, the chemical insecticide will always be able to eradicate the insects if the time between sprays is sufficiently short. However, as the period becomes longer the insecticide may not be able to eradicate the hosts even if large doses are applied each time. However, this is not true of the fungal pathogen where it is possible to eradicate the insects independently of the period of the spraying strategy. This is due to the fact that the fungal pathogens can inflict secondary infections via sporulation ($\omega > 0$), whilst chemical insecticides cannot ($\omega = 0$). If the pathogen is sufficiently virulent (i.e. β is sufficiently large) then the pathogen can eradicate the insects, independently of the spraying strategy adopted.

- With the inclusion of resistance, the differences between the chemical insecticide and the fungal pathogen is minimal. What is clear from the above analysis is that the underlying competition between the susceptible and resistant hosts determines the long-term behaviour. In particular, we see that both the fungal pathogen and the chemical insecticide fail to completely eradicate the insect population. However, by further understanding this intraspecific competition between hosts, advances may be made in developing partial control with either the pathogen or insecticide.

Chapter 4

Adaptive Dynamics

4.1 Motivation and Background

4.1.1 Chapter Outline

This chapter is on adaptive dynamics and the evolution of resistance. The model that is used is based upon the continuous time model presented in Chapter 2. Conditions are derived on the evolutionary outcomes that depend on the trade-off between susceptibility to infection from the fungal pathogen and intrinsic growth rates, and the amount of externally applied fungal pathogen. Moreover, I highlight the differences in evolutionary behaviour when differing forms of the carrying capacity are used.

4.1.2 Motivation

The theory of adaptive dynamics is used to explain the evolution of species, and in particular, it attempts to explain speciation through a process of evolutionary branching. The idea of adaptive dynamics is as follows:

- The individuals who constitute a population, the residents, will necessarily affect the environment they populate.
- New types of individuals, the mutants, arise at low density from small mutations.
- For the mutants to be successful they must first prosper in the environment determined by the resident.
- If the mutants are successful then they increase in density and begin to shape the environment.
- In the long-term the mutant may coexist with the original resident or oust it to become the new resident itself.

The theory imposes a clear separation of the (slow) evolutionary and (fast) population dynamical time scales, that is, mutations occur sufficiently infrequently that the population has reached its attractor before a new mutation occurs (Bowers & White 2002). With insect species this assumption may not strictly hold, especially when externally applied pathogens are used where evolution may act quickly. In Bowers, White, Boots, Geritz & Kisdi (2003) the authors allowed the new mutants to evolve before the previous mutants had reached equilibrium or gone extinct. This had no effect on the model predictions under the assumption that the mutants are allowed to go to equilibrium. Therefore, this rather strict assumption may be relaxed in some circumstances.
I will use this theory to explain the existence of resistant species of aphids.
As a motivational example I first consider the case where there is no fungal pathogen present. Then the population dynamics are modelled by the Lotka-Volterra competition model

$$\frac{dS}{dt} = r_S S\left(1 - \frac{S + \alpha_1 R}{K_S}\right) \tag{4.1a}$$

$$\frac{dR}{dt} = r_R R\left(1 - \frac{R + \alpha_2 S}{K_R}\right), \tag{4.1b}$$

where $S(t)$ and $R(t)$ are the densities of the susceptible and resistant aphids at time t respectively, r_i $(i = S, R)$ are the intrinsic growth rates, K_i $(i = S, R)$ are the carrying capacities and α_i $(i = 1, 2)$ are the competition coefficients. In this model I assume that the residents/susceptibles and mutants/resistants have the same competitive abilities so that $\alpha_1 = \alpha_2 = 1$.
Now let us assume that initially that the resident/susceptible population are at steady state $S^* = K_S$ (note this is the steady state for which the susceptible aphids approach when alone in the environment) and the mutant/resistant population is initially low. Then the mutant dynamics are well approximated by the differential equation

$$\frac{dR}{dt} = r_R\left(1 - \frac{S^*}{K_R}\right) R. \tag{4.2}$$

Hence it clearly follows that the mutant/resistant population can grow if, and only if,

$$r_R\left(1 - \frac{S^*}{K_R}\right) > 0, \tag{4.3}$$

or equivalently

$$K_R > S^* \quad \text{i.e.} \quad K_R > K_S. \tag{4.4}$$

This asserts that evolution will favour individuals capable of replacing themselves in the most crowded environment (Gurney & Nisbet 1998).

This simple example only suggests that in the absence of the pathogen, the aphids will evolve towards a state in which they have the largest steady state for the given environment. But of course this evolution of traits cannot go on without bound. There must be some physical or environmental factors that will limit the aphids' growth. This is one of the key ideas behind adaptive dynamics and trade-offs.

4.1.3 Background Reading

In Geritz, Kisdi, Meszéna & Metz (1998) the authors present a general framework for modelling adaptive trait dynamics in which are integrated various concepts and techniques from modern ESS-theory. An evolutionarily stable strategy (Maynard Smith & Price 1973) is defined as a strategy that cannot be displaced by any other known strategy, where strategy is defined as a behaviour (or set of behaviours) used by an individual to deal with an important life-history problem. The ESS is effectively defined as an evolutionary trap, with the main drawback being that it always remains to be seen whether during the course of evolution the ESS will actually become established at all. The authors also make the distinction between ESS-stability (which renders a population immune against invasion by any new mutant) and convergence stability (which ensures the gradual approach through a series of small evolutionary steps). The authors introduce the concept of an evolutionarily singular strategy as a generalisation of the ESS concept which is a point at which a number of evolutionary outcomes are possible.
The assumptions used for the framework are as follows:

- Individuals reproduce asexually, and offspring are phenotypically identical to the parent. Phenotypes are denoted by their strategy (which is assumed to be one-dimensional), which can vary continuously.
- The strategies in a given resident population can be considered as a set of model parameters that implicitly specify a unique attractor for the resident population dynamics.
- Mutations occur sufficiently infrequently so that the population has reached its attractor before a new mutant comes along.
- Each resident strategy present is protected against extinction by a positive growth rate when rare.
- The phenotypic mutations are small but random.

For monomorphic populations, fitness is defined as the long-term exponential growth rate of a phenotype in a given environment (see (4.2) and (4.3)) (Metz, Nisbet & Geritz 1992, Geritz et al. 1998). Sometimes this fitness is known as the marginal

growth rate of the rare mutant against the resident population (see Boots & Haraguchi (1999)).
Let E_x denote the environment in a population of a single phenotype x, and let $r(x, E_x)$ denote the population's long-term exponential growth rate. Hence, at the demographic attractor

$$r(x, E_x) = 0.$$

Now consider a new mutant with strategy y emerging in a population of residents with strategy x. Since the mutant is assumed to be rare, its effect on the environment E_x (set by the residents) is negligible. Therefore, the fitness of the mutant is equal to

$$\phi(y|x) = r(y, E_x).$$

If $\phi(y|x) > 0$ the mutant can spread, and if $\phi(y|x) < 0$ it will die out. If $\phi(y|x) > 0$ and $\phi(x|y) < 0$ then the mutant can spread but the resident cannot recover when rare itself, and therefore the mutant will normally eventually replace the resident.
Since the mutations are small, the authors are able to approximate the mutant's fitness by

$$\phi(y|x) = \phi(x|x) + D(x)(y - x)$$

which is in terms of the local fitness gradient, defined as

$$D(x) = \left.\frac{\partial \phi(y|x)}{\partial y}\right|_{y=x}.$$

However $\phi(x|x) = r(x, E_x) = 0$ for all x, and thus the sign of $D(x)$ determines which mutants can invade. The population evolves in the direction of the local fitness gradient until it reaches the neighbourhood of a strategy for which $D(x) = 0$. A strategy for which $D(x) = 0$ is called and evolutionarily singular strategy (or ESS).
The authors are then able to classify the properties of the singular strategies in terms of the fitness. The results of which can be found in Table 4.1 (note that the notation has been altered to coincide with the work that follows).
The four properties of the singular strategy are summarised below:

ESS-stable Otherwise known as evolutionarily unbeatable strategy (EUS) is if no nearby mutant can invade. A singular strategy that is ESS-stable is an evolutionary trap in the sense that once it has become established in a population, no further evolutionary change is possible by small mutations.

Convergence Stable A singular strategy that is convergence stable (CS) is an evolutionary attractor. A singular strategy that is not CS is an evolutionary repeller.

Singularity can Spread A singular strategy can spread in other populations when initially rare itself.

Mutually Invadable The strategy and any nearby strategy can mutually invade, and hence give rise to a dimorphic population.

Although these four properties are not fully independent of one another, the properties can be combined in various ways to yield eight different configurations, which exhibit different evolutionary behaviour.

In Bowers & White (2002) the authors marry together the theory of adaptive dynamics and trade-offs between evolving life-history constraints. As the authors point out, the mutation which occurs in adaptive dynamics probably will not affect just one parameter. What is becoming increasingly accepted is that a benefit gained in one area of a species life history (a model parameter) will trade-off with a cost in another. The key to this theory is the trade-off function, f, which links two parameters; one of which is the parameter that mutates, and the other which bears the cost.

An important study by Boots & Haraguchi (1999) considers the evolution of costly resistance in host-parasite systems, whereby the hosts interact with a non-evolving parasite. The authors concentrate on a trade-off between the susceptibility to infection and the intrinsic growth rate such that as the host becomes more resistant (i.e. less susceptible) to the pathogen, the host pays for the benefit by a reduced intrinsic growth rate. The model studied has the form

$$\begin{aligned} \frac{dX_i}{dt} &= r_i X_i - q\left(\sum_{i=0}^{n} X_i + Y\right) X_i - \beta_i X_i Y \\ \frac{dY}{dt} &= \sum_{i=0}^{n} \beta_i X_i Y - \Gamma Y \end{aligned}$$

where X_i is the density and r_i is the intrinsic growth rate of the i^{th} healthy strain of host; β_i is the rate at which the pathogen is transmitted to the i^{th} healthy strain of host; q is the coefficient of crowding which corresponds to r/K, where K is the carrying capacity; Y is the density of the infected class and Γ is the disease induced mortality rate. Notice that density dependence depends on the total population density of all the healthy strains and the infected. Hence, the model does not allow for varying intraspecific competition between strains.

With this model the authors are able to show that the evolutionary outcome depends crucially on the shape of the trade-off function between resistance and its assumed cost in intrinsic growth rates.

In Bowers et al. (2003) the authors use the theory of adaptive dynamics to highlight the differences in evolutionary behaviour when contrasting formulations of the carrying capacity are used. This notion will be more thoroughly discussed in the following

section.

4.2 The Adaptive Dynamics of Model (2.2) with Trade-Offs

An important question to ask is whether a mutant (more resistant) population of aphids can invade a resident population in the presence of a fungal pathogen?
In this section I will use the model considered in Chapter 2 to investigate the evolution of resistance in an aphid population. I shall do this in two ways; firstly I will consider the case where the carrying capacity is explicitly modelled, and secondly when the carrying capacity is implicitly modelled.

4.2.1 Model Formulation & Analysis

Carrying Capacity Explicitly Modelled

Consider a model for the resident (susceptible) population that interacts with a fungal pathogen. Its dynamics are given by (2.2) with $R \equiv 0$ and for notational convenience I shall write $\beta = \beta_S$ and $K_S = K$. Hence the model for the resident population is

$$\frac{dS}{dt} = r_S S\left(1 - \frac{S}{K}\right) - \beta_S SF = \left(r_S\left(1 - \frac{S}{K}\right) - \beta_S F\right) S \tag{4.5a}$$

$$\frac{dI}{dt} = \beta_S SF - \delta I \tag{4.5b}$$

$$\frac{dF}{dt} = \omega\delta I - dF + a. \tag{4.5c}$$

As seen in Chapter 2, Model 4.5 has a unique coexistence steady state (S^*, I^*, F^*), where $S^* < K$ and which is biologically realistic if, and only if,

$$a < \frac{dr_S}{\beta_S}.$$

In fact, this steady state is the resistants-eliminated steady state in the full model (2.2) with $a > 0$. I assume that this steady state is locally stable. I now consider a mutant (resistant) population with the same carrying capacity but with a different intrinsic growth rate, r_R, and transmission rate, β_R. Such trade-offs have been reported in experimental systems where species strains with a high resistance to disease pay a cost in terms of a reduced rate of disease-free reproduction (Boots & Begon 1993, Boots & Begon 1995). However, the precise correlation between the two is not clear.

Hence the full system is given by

$$\frac{dS}{dt} = r_S S\left(1-\frac{S+R}{K}\right) - \beta_S SF = \left(r_S\left(1-\frac{S+R}{K}\right) - \beta_S F\right) S \quad (4.6a)$$

$$\frac{dR}{dt} = r_R R\left(1-\frac{S+R}{K}\right) - \beta_R RF = \left(r_R\left(1-\frac{S+R}{K}\right) - \beta_R F\right) R \quad (4.6b)$$

$$\frac{dI}{dt} = \beta_S SF + \beta_R RF - \delta I \quad (4.6c)$$

$$\frac{dF}{dt} = \omega\delta I - dF + a. \quad (4.6d)$$

In this model it has been assumed that both S and R have the same interspecific competitive abilities.

I assume that the density of the mutants, R, is initially small. Thus the initial dynamics are

$$\frac{dR}{dt} = \left(r_R\left(1-\frac{S^*}{K}\right) - \beta_R F^*\right) R.$$

Hence the marginal growth rate of the rare mutant against the resident population is

$$\phi(r_R, \beta_R | r_S, \beta_S) = r_R\left(1-\frac{S^*}{K}\right) - \beta_R F^*.$$

This fitness function, ϕ, is key in adaptive dynamics. If ϕ is negative the mutant will die out, if ϕ is positive, the mutant may spread, increase in density and begin to interact with the environment.

I now assume that there exists a trade-off function between the intrinsic growth rate and transmissibility, such that

$$r_R = f(\beta_R) \quad \text{and} \quad r_S = f(\beta_S),$$

with $f' > 0$, which means that as the mutants become more resistant, they trade-off some of their ability to reproduce. Thus, using the equations for the steady state we get

$$\begin{aligned}\phi(\beta_R|\beta_S) &= f(\beta_R)\left(1-\frac{S^*(\beta_S)}{K}\right) - \beta_R F^* \\ &= \left(1-\frac{S^*}{K}\right)\left\{f(\beta_R) - \frac{\beta_R}{\beta_S} f(\beta_S)\right\},\end{aligned}$$

Property	Characteristic
Evolutionarily Unbeatable Strategy (EUS)	$\partial^2\phi/\partial\beta_R^2 < 0$
Convergence Stable (CS)	$\partial^2\phi/\partial\beta_S^2 - \partial^2\phi/\partial\beta_R^2 > 0$
Singularity can Spread (SPR)	$\partial^2\phi/\partial\beta_S^2 < 0$
Mutually Invadable (MI)	$\partial^2\phi/\partial\beta_S^2 + \partial^2\phi/\partial\beta_R^2 > 0$

Table 4.1: Properties of the evolutionarily singular strategy, β^* (see Geritz et al. (1998) and Bowers et al. (2003)). Note that all derivative are evaluated at $\beta_S = \beta_R = \beta^*$. Also note that EUS is sometimes also called ESS-stable.

since

$$\begin{aligned} F^* &= \frac{r_S}{\beta_S}\left(1 - \frac{S^*}{K}\right) \\ &= \frac{f(\beta_S)}{\beta_S}\left(1 - \frac{S^*(\beta_S)}{K}\right). \end{aligned}$$

I now analyse this model by using the method of adaptive dynamics (Geritz et al. 1998). Thus the mutants can invade if, and only if,

$$\phi(\beta_R|\beta_S) > 0 \quad \Leftrightarrow \quad \frac{f(\beta_R)}{\beta_R} > \frac{f(\beta_S)}{\beta_S}. \tag{4.7}$$

Hence any invading mutant that satisfies (4.7) will be able to invade the resident population. Given that mutations are small, the population will evolve along the fitness gradient $(\partial\phi/\partial\beta_R)$ until it reaches the neighbourhood of an evolutionarily singular strategy (ESS), β^*, which satisfies

$$\frac{\partial\phi}{\partial\beta_R}(\beta^*|\beta^*) = \left(1 - \frac{S^*(\beta^*)}{K}\right)\left\{f'(\beta^*) - \frac{f(\beta^*)}{\beta^*}\right\} = 0.$$

But since $S^* < K$, the candidate singular strategy must satisfy

$$f'(\beta^*) - \frac{f(\beta^*)}{\beta^*} = 0. \tag{4.8}$$

The behaviour at the singular strategy is determined from combinations of the non-mixed partial second derivatives of ϕ evaluated at $\beta_S = \beta_R = \beta^*$ and can be characterised by four properties (see Table 4.1).
Now,

$$\frac{\partial\phi}{\partial\beta_R}(\beta_R|\beta_S) = \left(1 - \frac{S^*(\beta_S)}{K}\right)\left\{f'(\beta_R) - \frac{f(\beta_S)}{\beta_S}\right\}$$

and so

$$\frac{\partial^2\phi}{\partial\beta_R^2}(\beta_R|\beta_S) = \left(1-\frac{S^*(\beta_S)}{K}\right) f''(\beta_R).$$

Also

$$\begin{aligned}\frac{\partial\phi}{\partial\beta_S}(\beta_R|\beta_S) &= -\frac{1}{K}\frac{\partial S^*(\beta_S)}{\partial\beta_S}\left\{f(\beta_R)-\frac{\beta_R}{\beta_S}f(\beta_S)\right\}\\ &\quad -\beta_R\left(1-\frac{S^*(\beta_S)}{K}\right)\left\{\frac{f'(\beta_S)}{\beta_S}-\frac{f(\beta_S)}{\beta_S^2}\right\},\end{aligned}$$

and therefore

$$\begin{aligned}\frac{\partial^2\phi}{\partial\beta_S^2}(\beta_R|\beta_S) &= -\frac{1}{K}\frac{\partial^2 S^*(\beta_S)}{\partial\beta_S^2}\left\{f(\beta_R)-\frac{\beta_R}{\beta_S}f(\beta_S)\right\}\\ &\quad +\frac{2\beta_R}{K\beta_S}\frac{\partial S^*(\beta_S)}{\partial\beta_S}\left\{f'(\beta_S)-\frac{f(\beta_S)}{\beta_S}\right\}\\ &\quad -\beta_R\left(1-\frac{S^*(\beta_S)}{K}\right)\left\{\frac{f''(\beta_S)}{\beta_S}-2\frac{f'(\beta_S)}{\beta_S^2}+2\frac{f(\beta_S)}{\beta_S^3}\right\}.\end{aligned}$$

Thus, evaluating the second derivatives of ϕ at the singular strategy we get

$$\begin{aligned}\frac{\partial^2\phi}{\partial\beta_R^2}(\beta^*|\beta^*) &= \left(1-\frac{S^*(\beta^*)}{K}\right) f''(\beta^*)\\ \frac{\partial^2\phi}{\partial\beta_S^2}(\beta^*|\beta^*) &= -\left(1-\frac{S^*(\beta^*)}{K}\right) f''(\beta^*)\end{aligned}$$

by (4.8).

According to Table 4.1, if the ESS is not EUS, then it cannot be CS, since if the ESS is not EUS then

$$\frac{\partial^2\phi}{\partial\beta_R^2}(\beta^*|\beta^*) > 0 \;\Leftrightarrow\; f''(\beta^*) > 0$$

and thus

$$\frac{\partial^2\phi}{\partial\beta_S^2}(\beta^*|\beta^*) - \frac{\partial^2\phi}{\partial\beta_R^2}(\beta^*|\beta^*) = -2\left(1-\frac{S^*(\beta^*)}{K}\right) f''(\beta^*) < 0,$$

that is, the ESS is not CS. Moreover, for this model EUS and CS are equivalent. Therefore this model is incapable of supporting branching/speciation, and only evolutionary attractor or repeller dynamics can be obtained. In fact, it is easy to see from (4.7) that any invading mutant that satisfies (4.7) will replace the resident. The population evolves to the type that maximises $f(\beta)/\beta$ and the unique global maximum of $f(\beta)/\beta$ (if it exists) is the global optimal strategy.

As the above analysis shows, this model can only exhibit attractor or repellor dynamics,

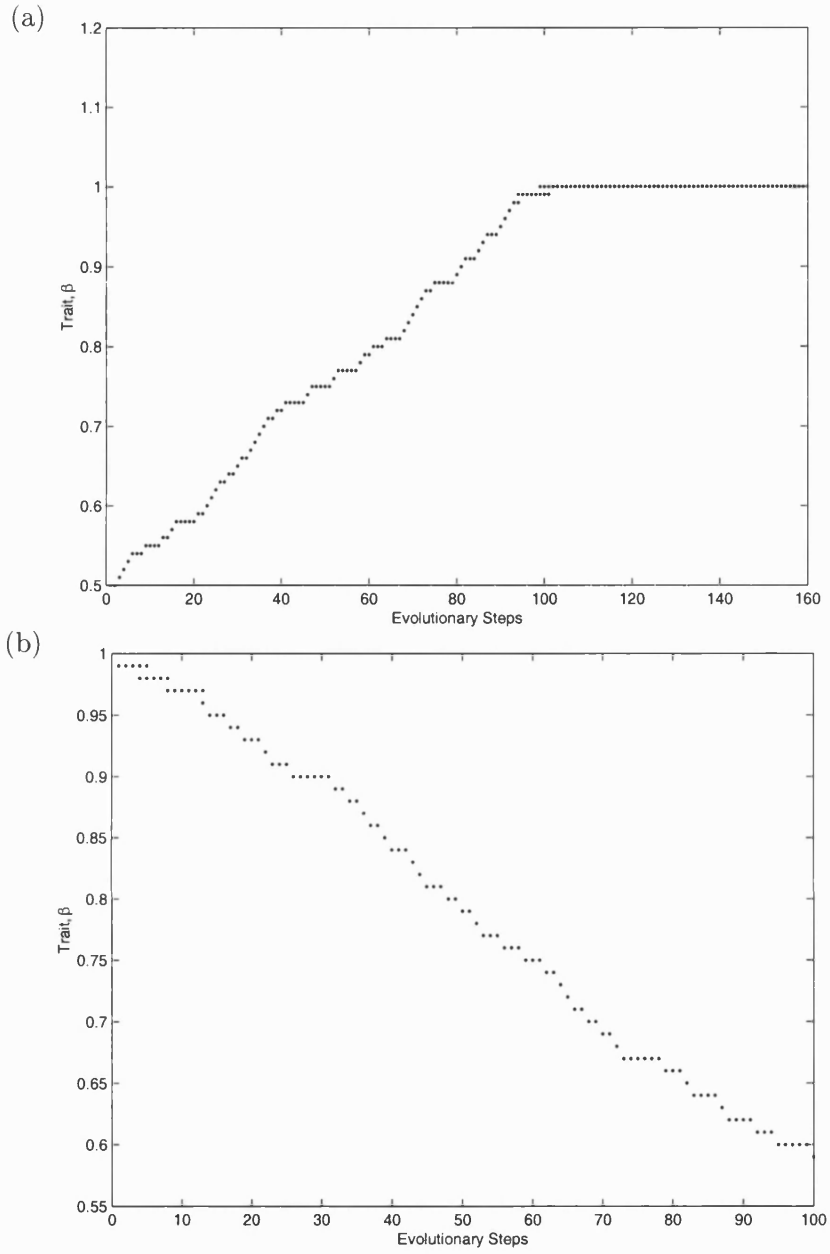


Figure 4-1: Two numerical simulations of Model 4.5. The model parameters for each plot are $K = 1$, $\delta = 0.1$, $\omega = 0.1$, $d = 1$, $a = 0.1$; and in (a) $\alpha = -0.3$ and so $\beta^* = 1$ is an evolutionary attractor, and in (b) $\alpha = 0.3$ and so $\beta^* = 1$ is an evolutionary repellor. See text for further details.

which is dependent on the sign of the second derivative of the trade-off function. In Figure 4-1 I use the trade-off function

$$f(\beta) = \alpha\beta^2 + 2\beta + \alpha. \tag{4.9}$$

Hence, the sign of the second derivative of f is given by the sign of α. Moreover, a candidate ESS, β^*, satisfies (4.8) and so using (4.9) the candidate ESS is $\beta^* = 1$. Note that for β values in the neighbourhood of β^* the trade-off function is positive for the values of α used in the numerical simulations. This is required so that the evolving intrinsic growth rates are always positive. Moreover, the parameters in the simulation satisfy all the criteria set out in Geritz et al. (1998) so that the technique may be used, i.e. we require $r = f(\beta) > 0$ and $f'(\beta) > 0$ for $\beta \approx \beta^*$ (see Figure 4-2).

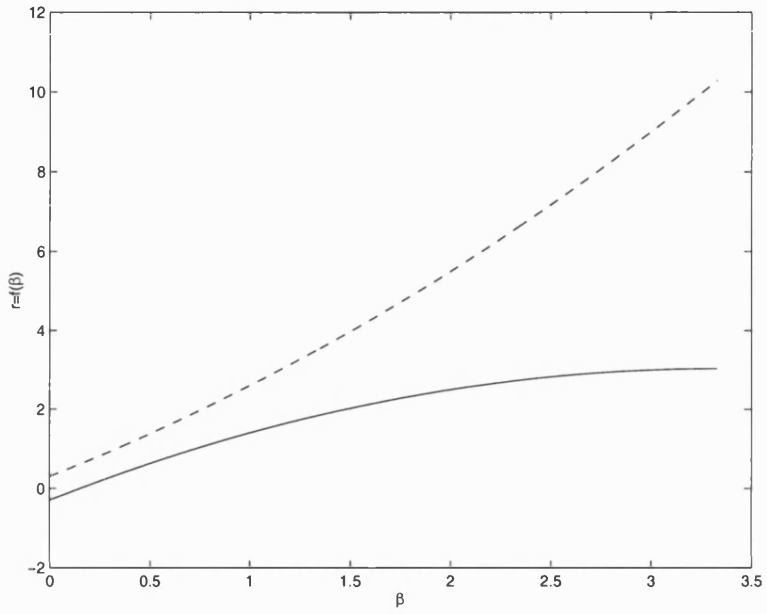


Figure 4-2: The function $r = f(\beta)$ for two values of α; $\alpha = -0.3$ the solid line; $\alpha = 0.3$ the dashed line.

The shape of the trade-off curves presented in this chapter are chosen so that the full range of behaviours can be observed. It is difficult and resource intensive to measure the shape of trade-off curves (Stearns 1992). However, it is possible to empirically model the trade-off curves by understanding the detailed mechanism of resistance. For example, a primary form of defence against pathogens in insects is passive barriers such as the gut wall and the cuticle, which effectively exclude the pathogen (Boots & Haraguchi 1999). I assume a model in which the resistance of the insect increases in proportion to the defence mechanism (such as an increased gut wall, cuticle or perhaps a encapsulation mechanism), in turn this must depend on the amount of resource available to the mechanism, and hence it must have a cost on another process of the insect such as the intrinsic reproductive rate (as assumed in my model).

This result is consistent with Bowers et al. (2003), where the authors considered a simpler predator-prey model. What is interesting about this result is that the singular strategy and its properties are determined only by the trade-off function and not the

parameter values of the insects or pathogen as one might expect. In particular, one might expect the amount of externally applied fungus will have some sort of influence on the evolutionary behaviour, whether this is in terms of the singular strategy value or the singular strategy's stability.

As Bowers et al. (2003) suggested, the explicit modelling of the carrying capacity can produce biologically counter-intuitive results. They also stated that an explicit carrying capacity may only be appropriate for systems where the major limiting resource is rigidly externally fixed, which is clearly not the case when considering aphids in the field. Therefore, they argued that there are good reasons to use an implicit formulation for the carrying capacity that is an emergent property of the intrinsic growth rate and a susceptibility to crowding.

Carrying Capacity Implicitly Modelled

I now consider the a marginally different model to (4.5), in that the carrying capacity is incorporated implicitly through a parameter q, the susceptibility to crowding. The model is

$$\frac{dS}{dt} = (r_S - qS - \beta_S F)\, S \tag{4.10a}$$

$$\frac{dI}{dt} = \beta_S SF - \delta I \tag{4.10b}$$

$$\frac{dF}{dt} = \omega\delta I - dF + a. \tag{4.10c}$$

The initially rare invading mutant dynamics are

$$\frac{dR}{dt} = (r_R - qS^* - \beta_R F^*)\, R,$$

where S^*, I^* and F^* are steady state solutions to (4.10) and are given by

$$S^* = \frac{B - \sqrt{B^2 - 4C}}{2} \tag{4.11a}$$

$$I^* = \frac{dF^* - a}{\omega\delta} \tag{4.11b}$$

$$F^* = \frac{r_S - qS^*}{\beta_S} \tag{4.11c}$$

where

$$B = \frac{r_S}{q} + \frac{d}{\omega\beta_S} \tag{4.11d}$$

$$C = \frac{dr_S}{q\omega\beta_S} - \frac{a}{q\omega}. \tag{4.11e}$$

I now introduce the trade-off function, $r_i = f(\beta_i)$ where $i = S, R$, and hence the fitness

function is given by

$$\begin{aligned}\phi(\beta_R|\beta_S) &= f(\beta_R) - qS^* - \beta_R F^*(\beta_S) \\ &= f(\beta_R) - f(\beta_S) - (\beta_R - \beta_S)F^*(\beta_S),\end{aligned}$$

since at equilibrium

$$f(\beta_S) - qS^* - \beta_S F^*(\beta_S) = 0. \tag{4.12}$$

Now, we have

$$\frac{\partial \phi}{\partial \beta_R}(\beta_R|\beta_S) = f'(\beta_R) - F^*(\beta_S),$$

and hence the singular points, β^* satisfy

$$\frac{\partial \phi}{\partial \beta_R}(\beta^*|\beta^*) = f'(\beta^*) - F^*(\beta^*) = 0, \tag{4.13}$$

where $F^*(\beta^*)$ is the equilibrium level of the non-trivial steady state of model (4.10). Now (4.13) is equivalent to

$$\beta^* f'(\beta^*) - f(\beta^*) + qS^*(\beta^*) = 0$$

by (4.11c). Then substituting (4.11a), (4.11d) and (4.11e), and simplifying we get that (4.13) is equivalent to

$$\left(\beta^* f'(\beta^*)\right)^2 - \beta^* f'(\beta^*)\left(f(\beta^*) - \frac{dq}{\omega\beta}\right) - \frac{aq}{\omega} = 0. \tag{4.14}$$

We also have

$$\frac{\partial^2 \phi}{\partial \beta_R^2}(\beta_R|\beta_S) = f''(\beta_R)$$

and

$$\frac{\partial \phi}{\partial \beta_S}(\beta_R|\beta_S) = -f'(\beta_S) + F^*(\beta_S) - (\beta_R - \beta_S)\frac{\partial F^*(\beta_S)}{\partial \beta_S}$$

and therefore

$$\frac{\partial^2 \phi}{\partial \beta_S^2}(\beta_R|\beta_S) = -f''(\beta_S) + 2\frac{\partial F^*(\beta_S)}{\partial \beta_S} - (\beta_R - \beta_S)\frac{\partial^2 F^*(\beta_S)}{\partial \beta_S^2}.$$

Hence, the non-mixed second derivatives at the singular point are

$$\begin{aligned}\frac{\partial^2\phi}{\partial\beta_R^2}(\beta^*|\beta^*) &= f''(\beta^*)\\ \frac{\partial^2\phi}{\partial\beta_S^2}(\beta^*|\beta^*) &= -f''(\beta^*)+2\frac{\partial F^*}{\partial\beta_S}(\beta^*)\\ &= -f''(\beta^*)-2\frac{q}{\beta^*}\frac{\partial S^*}{\partial\beta_S}(\beta^*)\end{aligned}$$

by (4.12) and (4.13).
Recall that the steady state, $S^* = S^*(\beta_S)$ is given by the quadratic

$$(S^*(\beta^*))^2 - BS^*(\beta^*) + C = 0,$$

where $B = B(\beta_S)$ and $C = C(\beta_S)$. Thus, it is simple to show that

$$\frac{\partial S^*}{\partial\beta_S}(\beta^*) = -\frac{f'(\beta^*)S^*(\beta^*)}{q\sqrt{(B(\beta^*))^2 - 4C(\beta^*)}},$$

and hence,

$$\frac{\partial^2\phi}{\partial\beta_S^2}(\beta^*|\beta^*) = -f''(\beta^*) + \frac{2f'(\beta^2)S^*(\beta^*)}{\beta^*\sqrt{(B(\beta^*))^2 - 4C(\beta^*)}}.$$

Thus, the singularity will be non-EUS if, and only if, $f''(\beta^*) > 0$ and convergence stable if, and only if,

$$f''(\beta^*) < \frac{f'(\beta^*)S^*(\beta^*)}{\beta^*\sqrt{(B(\beta^*)^2) - 4C(\beta^*)}} =: L.$$

Since $f' > 0$, it follows that branching/speciation occurs if, and only if,

$$0 < f''(\beta^*) < L. \tag{4.15}$$

To summarise, if $f''(\beta^*) < 0$, then β^* is an evolutionary attractor; if $f''(\beta^*) > 0$ but (4.15) is not satisfied then β^* is an evolutionary repeller, and if (4.15) is satisfied then β^* is an evolutionary branching point.

Numerical Simulations To investigate the types of behaviours observed I take the trade-off function to be

$$f(\beta) = \alpha\beta^2 + 2\beta + \alpha.$$

Substituting this trade-off function into (4.14) gives that the singular strategies are given by the roots of the quartic

$$\alpha^2\beta^4 + \alpha\beta^3 - \alpha^2\beta^2 + \alpha\left(\frac{qd}{\omega} - 1\right)\beta + \frac{q}{\omega}\left(d - \frac{a}{2}\right) = 0.$$

Now choosing suitable model parameter values one can find the all the singular strategies, and determine there evolutionary behaviour using the above analysis, or using a pairwise invasibility plot (or PIP) (see Kisdi & Meszéna (1995) and Metz et al. (1992) for examples).

$f''(\beta^*) > 0$: For $f''(\beta^*) > 0$ the above analysis tells us that if a singular strategy exists, then it is either an evolutionary branching point or an evolutionary repellor. Now, for the set of parameter values chosen for my simulations there are two biologically feasible singular strategies. These are shown in the PIP in Figure 4-3. In this a

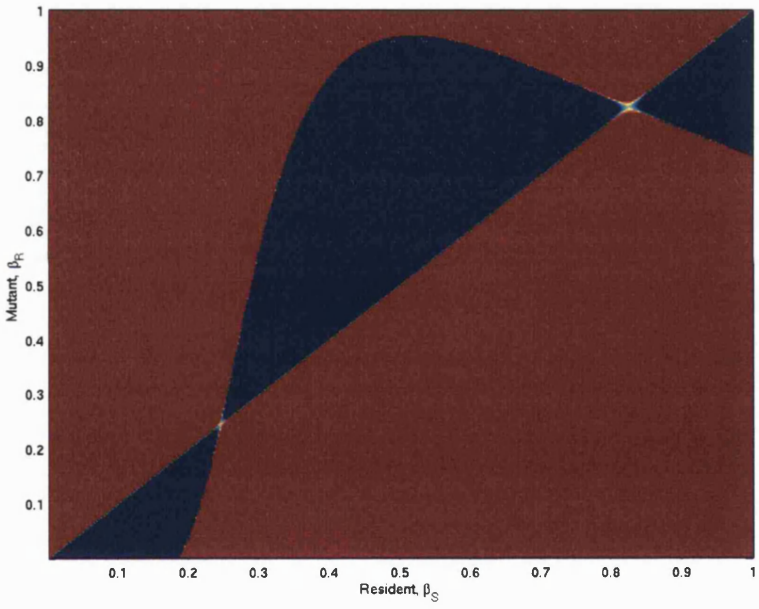


Figure 4-3: A pairwise invasibility plot for Model 4.10. The model parameters plot are $q = 0.1$, $\delta = 0.1$, $\omega = 0.1$, $d = 3.4$, $a = 5$ and $\alpha = 12$. Areas in red denote where $\phi(\beta_R|\beta_S) > 0$, areas in blue denote where $\phi(\beta_R|\beta_S) < 0$. See text for further details.

plot of the sign of $\phi(\beta_R|\beta_S)$ as a function of β_R and β_S. To see what mutants can spread in a given resident population, we look along a vertical line through a point on the x-axis representing the resident's strategy. The parts of this line inside a region that is red correspond to strategies on the y-axis for which $\phi(\beta_R|\beta_S) > 0$, and hence denote potentially invading mutants. The parts of the line inside a region that is blue correspond to mutants for which $\phi(\beta_R|\beta_S) < 0$, and therefore cannot invade. By

definition $\phi(\beta|\beta) = 0$, and the intersection of this diagonal with another line on which $\phi(\beta_R|\beta_S) = 0$ (i.e. the line which separates the red and blue regions) corresponds to a singular strategy, β^*. If mutations are small, we only need to consider strategies within a narrow band along the diagonal. A red region just above and a blue region just below the diagonal indicates a positive fitness gradient, whereas vice versa indicates a negative fitness gradient. Close to a singular strategy there are only eight possible generic local configurations of the PIP (Geritz et al. 1998).

For the parameters used in Figure 4-3 there are two singular strategies, $\beta_1^* \approx 0.245$ and $\beta_2^* \approx 0.824$. As the PIP indicates, β_1^* is a possible evolutionary branching point, and moreover $0 < f''(\beta_1^*) < L(\beta_1^*)$. Clearly, β_2^* is an evolutionary repellor, and moreover $0 < f''(\beta_2^*) \not< L(\beta_2^*)$. This result can be seen numerically in Figure 4-4 where we clearly see that β_2^* is an evolutionary repellor and β_1^* is an evolutionary branching point so that the initially monomorphic population evolves toward the singular strategy and then branches to a dimorphic population where both strains evolve away from the singular strategy in opposite directions.

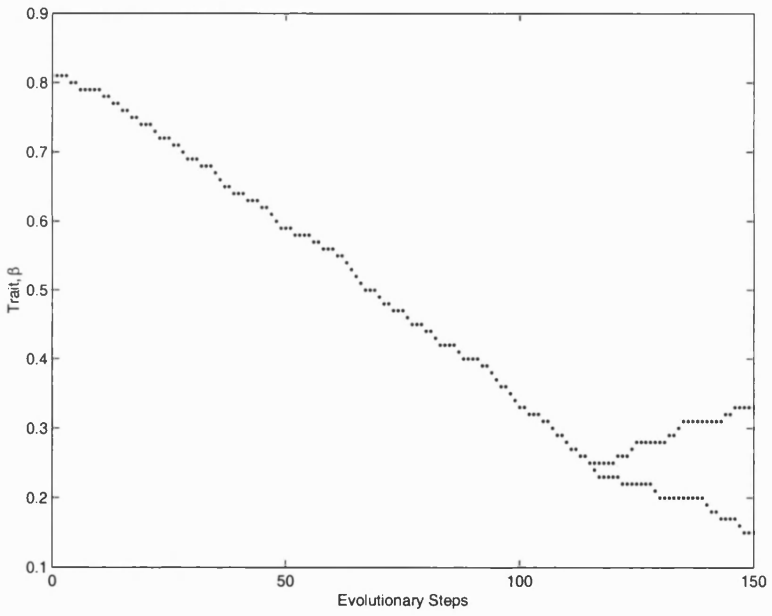


Figure 4-4: A simulation of Model 4.10. The model parameters are as in Figure 4-3.

$f''(\beta^*) < 0$: For $f''(\beta^*) < 0$ the above analysis tells us that if a singular strategy exists then it is an evolutionary attractor. In Figure 4-5 (a) the PIP indicates that there is a singular strategy at $\beta^* \approx 2.881$ and that it is an attractor. This is confirmed in Figure 4-5 (b) where the monomorphic population evolves towards the singular strategy and remains at the singular strategy once it has reached it.

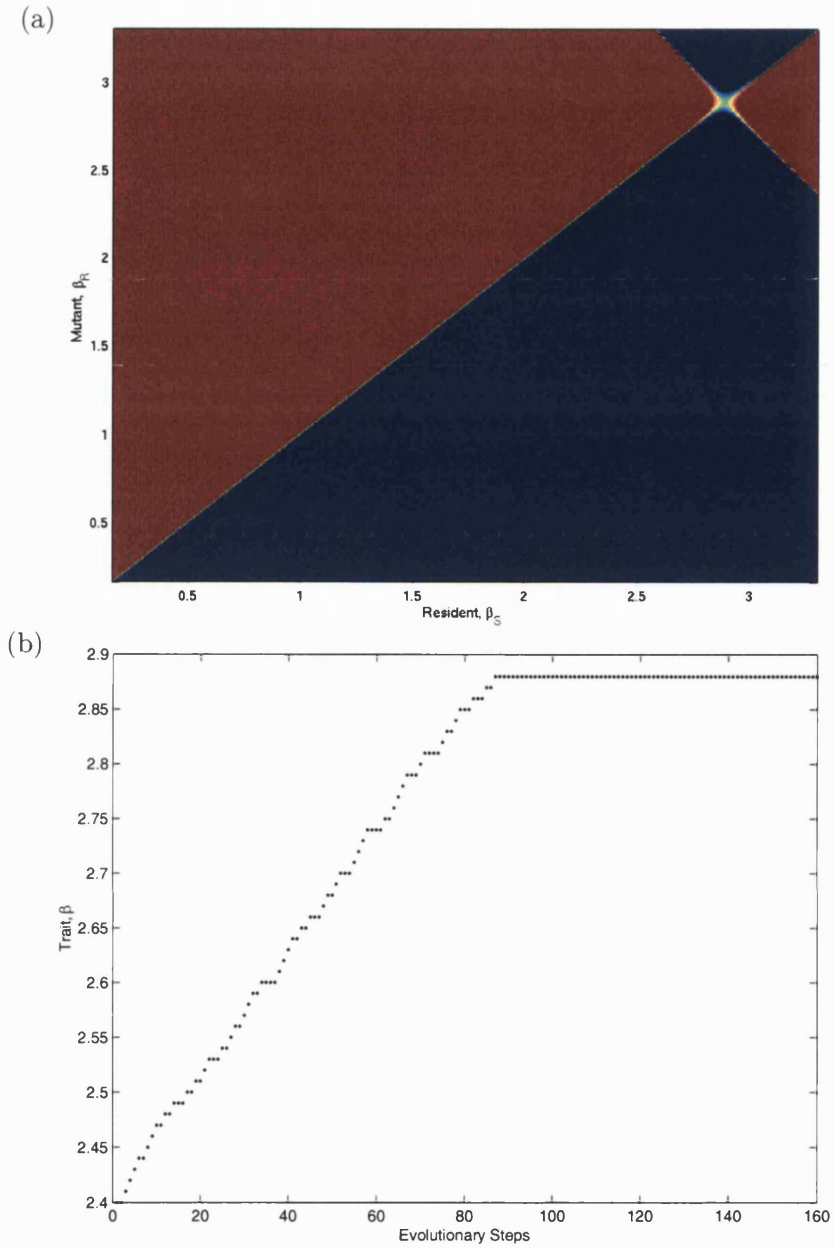


Figure 4-5: A PIP and a numerical simulation of Model 4.10. The model parameters for each plot are $q = 1$, $\delta = 0.1$, $\omega = 0.1$, $d = 1$, $a = 0.1$ and $\alpha = -0.3$. Since $\alpha < 0$ we may only consider a restricted interval of the resident and mutant strategies (see Figure 4-2).

The Evolutionary Dependence on the Application the of Pathogen: The question remains, 'How does a affect the evolution of the susceptibility trait in the insects?' Can we change a so that we can change the evolutionary outcome?

From the above analysis we know that if the carrying capacity is modelled explicitly then changing the amount of externally applied fungal pathogen, a, has no effect on

the evolutionary behaviour of the model which is unrealistic.
However, if the carrying capacity is implicitly modelled then the evolutionary outcome depends on the sign of the second derivative of the trade-off function, f. If $f''(\beta^*) < 0$ then the singular strategy (if it exists) is an evolutionary attractor. However, in this case the singular strategy depends on a. So by changing the amount of externally applied fungal pathogen can have an effect of the evolutionary attractor.

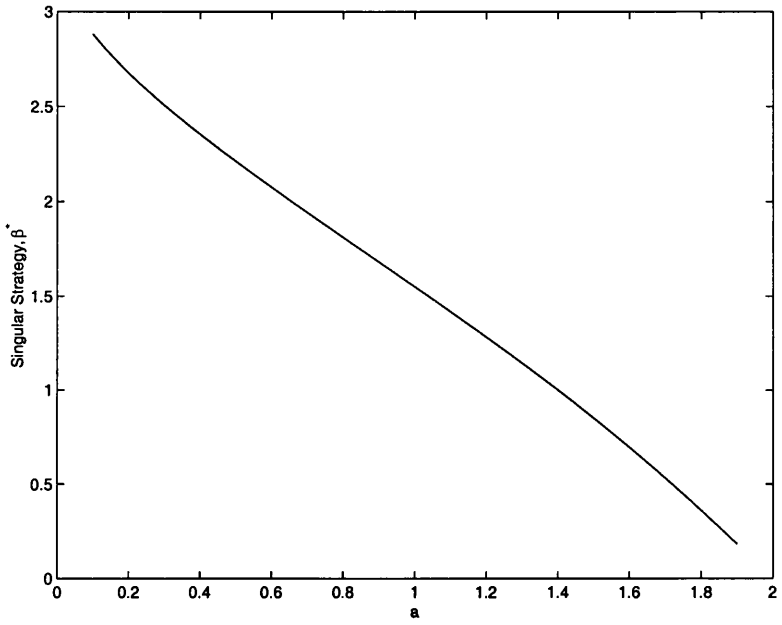


Figure 4-6: The effects on singular strategy, β^* as a changes. The model parameters are as in Figure 4-5.

In Figure 4-6 we see that as the amount of externally applied pathogen increases, the evolutionary attractor decreases. Thus, as more of the fungal pathogen is applied to the system, the insect species evolve towards a more resistant state. If a is sufficiently large then the singular strategy cannot exist, because if a is sufficiently large then no strain of insect will be able to coexist with the pathogen. Hence only completely resistant strains of insect will be able to survive in the environment.
If $f''(\beta^*) > 0$ then the singular strategy is either an evolutionary branching point or an evolutionary repellor. Thus varying a not only varies the singular strategy, but also has an effect on its stability.
In Figure 4-7 we see that there are at most three biologically realistic singular strategies for varying values of a. The singular strategy that corresponds to the black line denotes that the singular strategy satisfies (4.15) and so is a branching point. The strategies that correspond to a red line denotes that the singular strategy does not satisfy (4.15) and so the singular strategy is an evolutionary repellor. Hence for sufficiently low values of a if the populations initial strategy is low then the population will evolve towards the

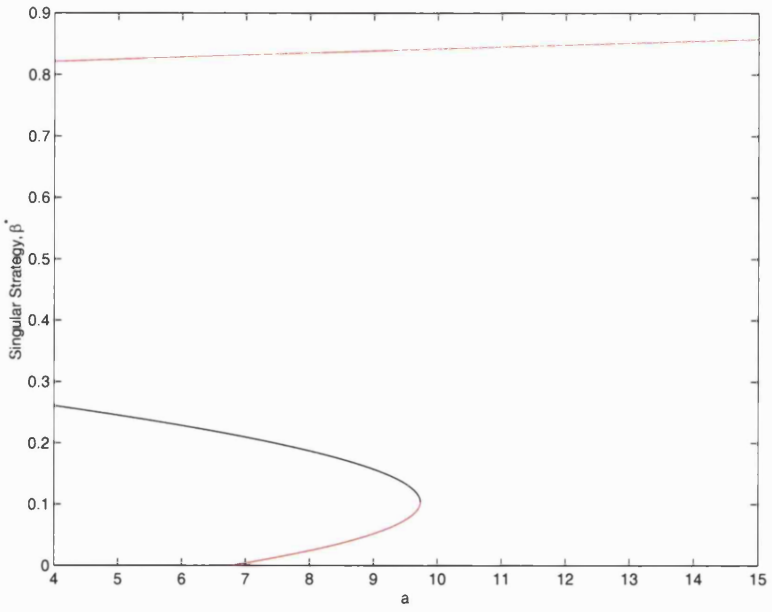


Figure 4-7: The effects on singular strategy, β^* as a changes. The model parameters are as in Figure 4-4. The black line denotes that the singular strategy is an evolutionary branching point. The red lines denote that the singular strategies are evolutionary repellors. See text for more detail.

branching singular strategy and the monomorphic population will become dimorphic, with one strain becoming more resistant whilst the other becomes more susceptible. However, if a is sufficiently large then the branching singular strategy does not exist, and so for an initially low in trait monomorphic population will remain monomorphic and evolve towards a more resistant state. Moreover, this becomes more likely as a increases the repelling singular strategy value increases.

4.3 Conclusions & Discussion

In this chapter I have demonstrated that branching/speciation is not possible when the carrying capacity is modelled explicitly, but is possible when the carrying capacity is modelled from the interplay between the intrinsic growth rate and a susceptibility to crowding. This may attributed to the fact that when the carrying capacity is modelled explicitly, the evolution does not change the carrying capacity, on the other hand, if the carrying capacity is modelled implicitly then the carrying capacity evolves as the population evolves.

When the carrying capacity is modelled explicitly there can only be evolutionary attraction or repulsion. Moreover, the singular strategy is independent of the host-pathogen parameter values. I believe that this seems to be biologically unrealistic in that one

can control the environment by varying the amounts of the fungal pathogen applied which one would expect to have some impact on the way in which the insects evolve.
If the carrying capacity is modelled implicitly we see a more complex set of evolutionary outcomes. In particular, the singular strategies and their stability depend on the amount of externally inputted fungal pathogen, with the consequence that is more pathogen is inputted into the environment, the more likely it is that the insects evolve towards a more resistant state, and in some cases may become completely resistant to the pathogen.
Throughout this chapter I have assumed that the trade-off is between susceptibility to infection and the intrinsic growth rate of the insect species. The form of this function has been chosen to satisfy certain criteria, for example $f' > 0$. However, I have no biological reasoning for choosing such a particular function, and to my knowledge there are no studies yet published to model this function. However, it is clear that the case where f is concave (i.e. $f'' < 0$) corresponds to the scenario where resistance becomes increasingly costly, and where f is convex (i.e. $f'' > 0$) corresponds to the scenario where resistance becomes decreasingly costly. Perhaps a biological reasoning for these two cases can be though of in the following way:

Increasingly Costly There are a limited amount of resources available to the development of the resistance mechanism. Therefore, to become more resistant to the pathogen the insect species must pay a higher and higher price in terms of its reproductive output.

Decreasingly Costly If the costly defence mechanism is produced, but once produced, has a wide range of defence at little extra cost then this would make the resistance decreasingly costly.

Boots & Haraguchi (1999) considered a trade-off function which is sigmoidal in shape, since it is unlikely that the resistance mechanism becomes indefinitely cheaper to produce as in the decreasingly costly case (f convex). The sigmoidal curve assumes that after a region in which the trade-off function is decreasingly costly, the trade-off reaches maximal efficiency, after which becomes increasingly costly. However, the authors found that this curve effectively had the same properties as the more simple convex (decreasingly costly) trade-off function.
Clearly further studies are required to better understand the trade-offs associated with resistance to fungal pathogens, so that work similar to that presented in this chapter can be extended to incorporate more complex (and perhaps more realistic) dynamics.

Chapter 5

Discrete-Time Model

5.1 Motivation and Background

5.1.1 Chapter Outline

In this chapter I present a discrete-time insect-pathogen model to describe the interaction between a host insect that can develop a resistant strain, and a fungal pathogen that can be applied externally to the system. I then investigate the long-term behaviour of the model for three scenarios, when no pathogen is present, when there is no externally applied pathogen, and when the pathogen is externally applied. These three scenarios are then compared.

5.1.2 Motivation

Many insect species have non-overlapping generations or are subject to some naturally occurring periodicity within their life-cycles. Aphids have essentially two modes of over-wintering: holocylic species produce a sexual generation in autumn, which mate to produce over-wintering eggs. Anholocylic species are dependent on parthenogenic females surviving the winter (Wade & Leather 2002). A diagrammatic representation of a holocylic species is presented in Figure 5-1, which is considered in this chapter.

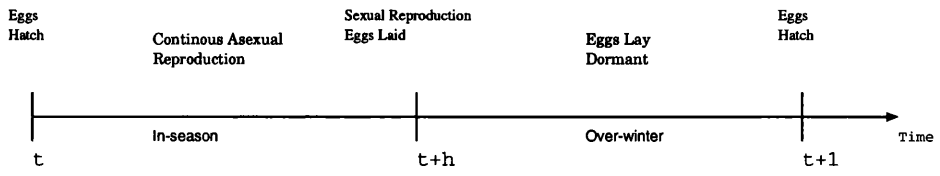


Figure 5-1: Aphid Life-Cycle.

To model this type of naturally occurring periodicity it is often convenient to use a discrete-time model. In the background reading section that follows I discuss some

models that use discrete time.

5.1.3 Background Reading

I begin with the classical Nicholson-Bailey model (Nicholson & Bailey 1935). The model describes a host-parasitoid interaction, and assumes that the census takes place at the beginning of the season, before parasitism takes place. The model has the form

$$\begin{aligned} N_{t+1} &= rN_t f(N_t, P_t) \\ P_{t+1} &= \omega N_t \left(1 - f(N_t, P_t)\right) \end{aligned}$$

where

- N_t is the number (or density) of hosts at generation t;
- P_t is the number of parasitoids at generation t;
- r is the per capita reproduction of the host in the absence of parasitism (also known as the basic reproductive ratio);
- ω is the average number of parasitoids emerging from a single parasitised host;
- $f(N_t, P_t)$ is the fraction of hosts that escape parasitism.

One major criticism of the model is that in the absence of the parasitoids, the hosts grow exponentially if the basic reproductive ratio is greater than unity. The reason for this is that the model does not allow for host density dependent effects such as host intraspecific competition. Hence if any host population regulation exists it must come from the parasitoids.
Classically the the parasitoid search for a host is assumed to be a Poisson process which leads to the function f taking the form

$$f(N_t, P_t) = \exp(-\beta P_t)$$

where β is known as the searching efficiency.
With this form of f, the Nicholson-Bailey model has unrealistic behaviour in that as time increases both the hosts and parasitoids exhibit oscillations which grow in amplitude without bound (see Murray (1989) or Britton (2003) for further details).
Other forms of f have been considered by many authors (Rogers 1972, Beddington 1975, May 1978, Hassell 1980) who consider the interactions between the parasite and the host in more detail. In particular, theorists have considered interactions where the parasitoids search according to a distribution which is more clumped than a Poisson

distribution. For example May (1978) considered f to take the form

$$f(N_t, P_t) = \left(1 + \frac{\beta P_t}{k}\right)^{-k}$$

where k is the exponent from the negative binomial distribution and is often known as the clumping parameter. The use of this form of f can stabilise the Nicholson-Bailey model if $k < 1$. Other biological factors that have been considered include the amount of time wasted by the parasitoid when encountering a host, the total time of the interaction and handling time.

As mentioned above, in the absence of parasitoids in the Nicholson-Bailey model the hosts dynamics are density independent where the model has the form

$$N_{t+1} = F(N_t) \quad \text{where} \quad F(N_t) = rN_t.$$

Many authors have investigated different forms of $F(N_t)$ which are nonlinear to incorporate intraspecific competition. A list of such functions can be found in May & Oster (1976). Two of the more commonly used forms are the Ricker model (Ricker 1954) (or sometimes it is known as the Moran-Ricker equation (Moran 1950)) which has the form

$$F(N_t) = N_t \exp\left[r\left(1 - \frac{N_t}{K}\right)\right]$$

and the Hassell model (Hassell 1974) which has the form

$$F(N_t) = \frac{\lambda N_t}{(1 + aN_t)^b}.$$

Both of these models exhibit stable or unstable dynamics depending on the parameter space.

Although there is a wealth of publications on host-parasite models, there are relatively few that deal with host resistance to the parasite. However White & Wilson (1999) consider a discrete-time model where the host population is split into two categories; those who are susceptible to a pathogen (which can be though of as a type of parasite) and those who are resistant. The model considered has the form

$$\begin{aligned} S_{t+1} &= \Lambda_S f(N_t) N_t \\ R_{t+1} &= \Lambda_R (1 - f(N_t)) N_t \\ P_{t+1} &= \sigma_P P_t + \lambda(\sigma_S(0) - \sigma_S(P_i)) S_t \end{aligned}$$

where

- S_t, R_t and P_t are the densities of susceptible larvae, resistant larvae and free-living pathogen at the start of the t^{th} generation respectively;
- $N_t = \sigma_S(P_t)S_t + \sigma_R R_t$ is the total number of hosts which survive to the end of the t^{th} generation;
- Λ_i is the basic reproductive ratio;
- λ is the number of pathogen propagules produced per infected death;
- $f(N_t)$ is the fraction of surviving individuals giving birth to susceptibles;
- σ_i is the survival probability.

The authors show that the inclusion of a resistant class can stabilise unstable host-pathogen interactions but there is greatest regulation when the fraction born resistant is density independent. Nonetheless, inclusion of density dependence can still allow intrinsically unstable host-pathogen dynamics to be stabilised provided that this effect is sufficiently small.
Although this model takes into account the dynamics of resistance it does not deal with the application of the pathogen to the system, which is important in controlling the pest host species. In the model developed in this chapter I take into account the application of the pathogen and how it affects the susceptible and resistant hosts.

5.2 Model Formulation

For the model presented in this chapter, I make use of the fact that aphids can have non-overlapping generations and use a discrete-time population model which is based on the Nicholson-Bailey model. I assume that for the host population

- there are two subclasses of host which have a total density N_t at generation t, where one subclass is susceptible to the pathogen and the other is totally resistant to the pathogen;
- the average number of offspring produced by surviving individuals (basic reproductive ratio) is λ_i, where $i = 1$ is the susceptible subclass and $i = 2$ is the resistant subclass;
- the probability that an individual from subclass i survives the generation is assumed to be density dependent and of the form $d_i e^{-r_i N_t}$, where d_i is the probability of survival in the absence of density dependent effects and r_i is the coefficient of density dependence;
- the probability of remaining susceptible at each generation is p, and thus $1-p$ is the probability of becoming resistant.

Thus the host dynamics in the absence of the pathogen is given as

$$N_{t+1} = \lambda_1 d_1 p N_t e^{-r_1 N_t} + \lambda_2 d_2 (1-p) N_t e^{-r_2 N_t}. \tag{5.1}$$

Let Φ_t be the density of free-living pathogen at generation t and assume that

- the probability that a susceptible individual avoids pathogenesis is $f(\Phi_t)$, where I assume that the process is Poisson with mean $\beta\Phi_t$, so that $f(\Phi_t) = e^{-\beta\Phi_t}$;
- the average amount of pathogen released from an infected susceptible host is ω;
- the average proportion of pathogen surviving to the next generation is $\alpha \in [0, 1)$;
- the average amount of pathogen applied externally to the environment at each generation is a. I interpret this parameter as a potential control mechanism for the hosts.

For notational convenience I introduce the parameter $A_i = \lambda_i d_i$ ($i = 1$, susceptible; $i = 2$, resistant) which denotes the average contribution by each subclass to the next generation in the absence of density dependent effects or pathogenesis. Hence under the above assumptions the full model system is

$$N_{t+1} = A_1 p N_t e^{-r_1 N_t} f(\Phi_t) + A_2 (1-p) N_t e^{-r_2 N_t} \tag{5.2a}$$

$$\Phi_{t+1} = \omega p N_t (1 - f(\Phi_t)) + \alpha\Phi_t + a. \tag{5.2b}$$

Notice that the host population has density dependence acting upon it through survival which is independent of the pathogen presence, hence the pathogen impact is independent of that process. Moreover, the density dependence on the host population impacts on the pathogen by lowering N_t in the next generation.
This model also assumes that if a resistant host is encountered by a free-living pathogen then the resistant host does not become infected and hence no pathogen is released upon the resistant hosts death. Thus if the resistant host densities are relatively high (i.e. p is relatively small) then this will have an impact on the pathogen levels.
The model also assumes that p is constant. This assumption is made so that the effects of the external spraying strategy, a, and the role of α can be clearly explored. The analysis of the model is carried out in several stages, exploring in particular the long-term outcomes. Initially, the interaction between susceptible and resistant subclasses was analysed in the absence of the pathogen ($\hat{N} > 0$, $\hat{\beta} = 0$). This is done to understand the underlying behaviour of the host dynamics, so that the presence of the pathogen can analysed. Following this a "natural" pathogen was introduced into the system ($\beta > 0$, $a = 0$) and finally the complete model system was considered. This allows comparisons between a controlled and uncontrolled system. The results are discussed below.

5.3 Results

Throughout this section, $\hat{N}$ refers to the host only non-trivial steady state ($\Phi \equiv 0$), and N^* refers to the host-pathogen non-trivial steady state ($N^*, \Phi^* > 0$).
I begin with examining the case where no pathogen is present.

5.3.1 No Pathogen

Setting $\beta = 0$ in (5.2), I consider the case when no pathogen interacts with the hosts. Thus we have the reduced model

$$N_{t+1} = A_1 p N_t e^{-r_1 N_t} + A_2(1-p)N_t e^{-r_2 N_t} =: F(N_t). \tag{5.3}$$

For ease of reading I now investigate the long-term behaviour of (5.3) in the following lemma.

Lemma 5.3.1. *Model 5.3 has two steady states; the trivial steady state, $N = 0$, which is linearly stable if, and only if,*

$$A_1 p + A_2(1-p) < 1, \tag{5.4}$$

and the non-trivial host-only steady state $N = \hat{N} > 0$ which exists if, and only if,

$$A_1 p + A_2(1-p) > 1. \tag{5.5}$$

Moreover, it is impossible for the steady state to bifurcate through a tangent bifurcation (+1 bifurcation) for all parameter space, and if $A_1 p A_2(1-p) > e^2$ then $\hat{N}$ is not linearly stable.

Proof. Setting $N_{t+1} = N_t = N$ in (5.3) we get that $N = 0$ or

$$1 = A_1 p e^{-r_1 N} + A_2(1-p)e^{-r_2 N}. \tag{5.6}$$

Now let

$$f(N) = A_1 p e^{-r_1 N} \quad \text{and} \quad g(N) = 1 - A_2(1-p)e^{-r_2 N}.$$

Now $f(0) = A_1 p > 0$, $f(N) \to 0$ as $N \to \infty$ and $f(N) > 0$ for all $N > 0$. Also $g(0) = 1 - A_2(1-p)$ and $g(N) \to 1$ as $N \to \infty$. Then by the monotonicity of f and g (since $f' < 0$ and $g' > 0$ $\forall N$), there exists a unique positive equilibrium $\hat{N}$ given by $f(\hat{N}) = g(\hat{N})$ if, and only if, (5.5) holds.
To determine the stability of the equilibria we see if $|F'(N)| < 1$ at the steady state, where

$$F'(N) = (1 - r_1 N)A_1 p e^{-r_1 N} + (1 - r_2 N)A_2(1-p)e^{-r_2 N}.$$

Thus the trivial steady state, $N = 0$, is stable if, and only if, $A_1 p + A_2(1-p) < 1$ and the non-trivial steady state, $N = \hat{N}$ is stable if, and only if,

$$0 < r_1 A_1 p \hat{N} e^{-r_1 \hat{N}} + r_2 A_2 (1-p) \hat{N} e^{-r_2 \hat{N}} < 2$$

by (5.6). Hence the non-trivial steady state is stable if, and only if,

$$r_1 A_1 p \hat{N} e^{-r_1 \hat{N}} + r_2 A_2 (1-p) \hat{N} e^{-r_2 \hat{N}} < 2. \tag{5.7}$$

Moreover, we see that it is impossible for the steady state to bifurcate through a tangent bifurcation (+1 bifurcation).
By (5.6)

$$\hat{N} > \frac{\ln(A_1 p)}{r_1} \quad \text{and} \quad \hat{N} > \frac{\ln(A_2(1-p))}{r_2}$$

and therefore

$$r_1 A_1 p \hat{N} e^{-r_1 \hat{N}} + r_2 A_2 (1-p) \hat{N} e^{-r_2 \hat{N}} > \ln(A_1 p) + \ln(A_2(1-p)). \tag{5.8}$$

Thus if $A_1 p A_2 (1-p) > e^2$ then $\hat{N}$ is not linearly stable. □

Condition (5.5) can be expressed as the joint average contribution to the next generation exceeding unity, which means biologically that the population will grow exponentially in the absence of density dependent effects. This condition features throughout this chapter.
Lemma 5.3.1 shows that it is impossible to have a tangent bifurcation at the non-trivial steady state, but numerics suggest that the steady state can be destabilised via a pitchfork bifurcation (see Figure 5-2), which occurs when inequality (5.7) is violated. This type of complex dynamics is typical for this type of discrete-time model (May & Oster 1976).

The Role of p

The host only non-trivial steady state exists if, and only if, (5.5) holds. In Table 5.1 we demonstrate how p affects the validity of this inequality in terms of the average contributions to the next generation from each subclass. In particular, if both A_1 and A_2 are less than unity then the non-trivial steady state does not exist. This is consistent with the extreme cases (i.e. when $p = 0$ or $p = 1$) in which the average contribution to the next generation of each subclass must exceed unity for the steady state to exist. At the other extreme, if each subclass has an average contribution to the next generation larger than unity, then any proportion of susceptible and resistant individuals will result in a non-trivial total population. In the remaining cases p is restricted to either

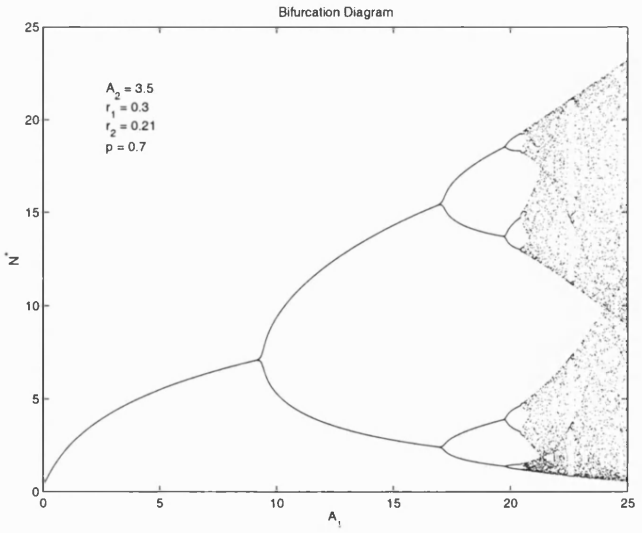


Figure 5-2: Bifurcation diagram on the no pathogen model (5.3).

	$A_2 < 1$	$A_2 > 1$
$A_1 > 1$	$p > p^*$	Any p
$A_1 < 1$	No p	$p < p^*$

Table 5.1: Conditions on the average contributions from each subclass (either susceptible or resistant) and p such that there exists a positive equilibrium to Model 5.3. $p^* = \frac{1-A_2}{A_1-A_2}$.

$0 < p < p^*$ or $p^* < p < 1$ where $p^* = (1 - A_2)/(A_1 - A_2)$. This result means that the subclass (either the susceptible or resistant) which has exponential growth in the absence of density dependent effects needs to make up a sufficiently large proportion of the whole population.

Model Simplification

It is not possible to find an explicit expression for the host-only non-trivial steady state, $\hat{N}$ from (5.6) or to determine tractable linear stability criteria. Here I consider two simplifications which allow us to obtain approximate solutions for the non-trivial steady state and its stability. These are then used to validate the numerical results obtained for the no pathogen model and the complete system.

$\mathbf{r_2 \approx r_1}$: If $r_2 = r_1$, the model system (5.3) simplifies to the Ricker equation with non-trivial equilibrium

$$\hat{N} = \frac{\ln(A_1 p + A_2(1-p))}{r_1}, \tag{5.9}$$

which has stability condition

$$1 < A_1 p + A_2(1-p) < e^2. \tag{5.10}$$

Now suppose that $r_2 \approx r_1$, and set $r_2 = r_1(1+\varepsilon)$ where $|\varepsilon| \ll 1$. This leads to a regular perturbation problem to solve for the steady state (5.6). Taking $\hat{N} = N_0 + \varepsilon N_1$ to $O(\varepsilon)$ and substituting into (5.6) gives

$$N_0 = \frac{\ln(A_1 p + A_2(1-p))}{r_1} \quad \text{and} \quad N_1 = -N_0 \frac{A_2(1-p)}{A_1 p + A_2(1-p)}. \tag{5.11}$$

Note that as $\varepsilon < 0$ decreases from zero, the non-trivial steady state increases, and conversely, as $\varepsilon > 0$ increases from zero, the non-trivial steady state decreases. This is intuitively obvious since a decrease in the density dependent effects should increase the population steady state level.

Considering the linear stability condition for this non-trivial steady state, to $O(\varepsilon)$ the stability condition is the same as the case when $r_2 = r_1$. Thus if the combined contribution to the next generation is too large ($A_1 p + A_2(1-p) \gtrapprox e^2$), population outbreaks can be observed.

$\mathbf{r_2 \ll r_1}$: Setting $r_2 = \varepsilon r_1$ where $0 < \varepsilon \ll 1$, I obtain a perturbation solution to (5.6) which to $O(\varepsilon)$ is

$$\hat{N} = N_0 + \varepsilon N_1 \tag{5.12}$$

where

$$N_0 = \frac{1}{r_1} \ln\left(\frac{A_1 p}{1 - A_2(1-p)}\right) \quad \text{and} \quad N_1 = -\frac{A_2(1-p)}{1 - A_2(1-p)} N_0.$$

I must impose $A_2(1-p) < 1$ otherwise N_0 is undefined and (5.5) must hold for the steady state to be biologically realistic. This is clearly shown in the limiting case where $\varepsilon = 0$ (i.e. $r_2 = 0$), where the host population will grow exponentially if $A_2(1-p) > 1$ and host only steady state does not exist. Taking $\varepsilon > 0$, increases the density dependent effects of the resistant population, and hence decreases the total host population steady state.

For stability the $O(1)$ condition is

$$\frac{A_1 p}{1 - A_2(1-p)} < \exp\left(\frac{2}{1 - A_2(1-p)}\right). \tag{5.13}$$

In Table 5.2 I explore the effect p has on the existence and stability of the $O(1)$ non-trivial steady state in (5.12), which is equivalent to setting $r_2 = 0$ in (5.3). Biologically this means that there is no density dependent effects acting on the

	$A_1 < 1$	$1 < A_1 < e^2$	$A_1 > e^2$
$A_2 < 1$	No p	$p\epsilon(\hat{p}, 1]$	$p\epsilon(\hat{p}, p^*)$
$A_2 > 1$	$p\epsilon(p^*, \hat{p})$	$p\epsilon(p^*, 1]$	$p\epsilon(\tilde{p}, p^*)$

Table 5.2: Conditions on p such that the $O(1)$ non-trivial steady state in (5.12) is biologically realistic and stable. $p^*\epsilon(0,1)$ is given by the solution to the implicit equation $\frac{A_1 p^*}{1-A_2(1-p^*)} = \exp\left(\frac{2}{1-A_2(1-p^*)}\right)$, $\hat{p} = \frac{A_2-1}{A_2-A_1}$ and $\tilde{p} = \frac{A_2-1}{A_2} < p^*$.

resistant subclass.

- If $A_1 < 1$ then in the absence of the resistant subclass ($p = 1$) the host population would die out, i.e. no non-trivial steady state would exist. However, if $A_2 > 1$ (i.e. the average contribution to the next generation in the resistant subclass is greater than unity) then a stable non-trivial steady state is possible for a specific range of p. What is interesting about this result, is that in the absence of susceptible class ($p = 0$) the host population would grow exponentially.
- If $1 < A_1 < e^2$ then in the absence of the resistant subclass the host population would exhibit a stable non-trivial steady state. Hence, the host population retains a stable non-trivial steady state with the introduction of the resistant subclass if p is sufficiently large.
- If $A_1 > e^2$ then in the absence of the resistant subclass the host population would exhibit oscillatory behaviour. However, introducing sufficient proportions of the resistant subclass can have a stabilising effect.

In Figure 5-3 I present numerical simulations of Model 5.3 when $r_2 \approx r_1$ and $r_2 \ll r_1$. The approximate solution always underestimates the true solution for ε sufficiently small since the $O(\varepsilon^2)$ correction term is always positive.

5.3.2 No Externally Applied Pathogen

Setting $a = 0$ in (5.2) gives rise to a model system which describes host-pathogen interactions in the absence of external applications of the pathogen. The control strategy is to apply an initial application of pathogen and rely on its pathogenicity to regulate the host population levels in subsequent generations.

Lemma 5.3.2. *The steady state solutions in this case are given as*

$$(N, \Phi) = (0,0), (\hat{N}, 0), (N^*, \Phi^*) \tag{5.14}$$

where $\hat{N}$ satisfies (5.6)*, and the non-trivial steady state $(N, \Phi) = (N^*, \Phi^*)$ satisfies the*

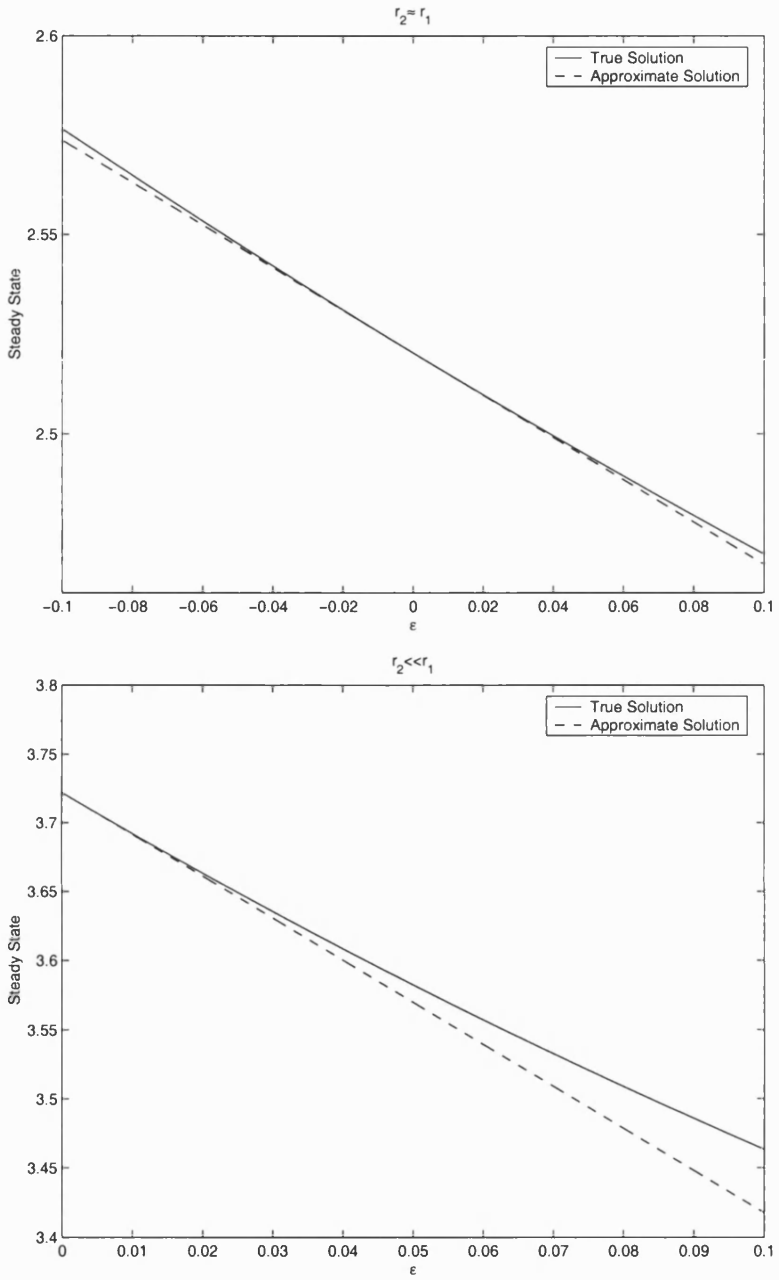


Figure 5-3: Numerical and approximate non-trivial steady state solutions to Model 5.3. The model parameters are $A_1 = 2.4, A_2 = 1.5, p = 0.7$ and $r_1 = 0.3$.

implicit relation $f(N^*) = g(N^*)$ *where*

$$f(N^*) = \frac{1-\alpha}{\beta} \ln\left(\frac{A_1 p e^{-r_1 N^*}}{1 - A_2(1-p)e^{-r_2 N^*}}\right), \tag{5.15a}$$

$$g(N^*) = \omega p N^* \left(1 - \frac{1 - A_2(1-p)e^{-r_2 N^*}}{A_1 p e^{-r_1 N^*}}\right), \tag{5.15b}$$

$$\Phi^* = \frac{f(N^*)}{1-\alpha}. \tag{5.15c}$$

This relation admits a biologically realistic host-pathogen non-trivial steady state solution $(N, \Phi) = (N^*, \Phi^*)$ *if*

$$A_1 p + A_2(1-p) > 1 \quad \textit{and} \quad \hat{N} > \frac{1-\alpha}{\omega\beta p}. \tag{5.16}$$

Proof. Setting $N_{t+1} = N_t = N$ and $\Phi_{t+1} = \Phi_t = \Phi$, the non-trivial steady state equations are

$$\begin{aligned} 1 &= A_1 p e^{-r_1 N} e^{-\beta\Phi} + A_2(1-p)e^{-r_2 N} \\ \Phi &= \omega p N\left(1 - e^{-\beta\Phi}\right) + \alpha\Phi. \end{aligned}$$

Therefore,

$$0 < \frac{1 - A_2(1-p)e^{-r_2 N}}{A_1 p e^{-r_1 N}} = e^{-\beta\Phi} < 1 \tag{5.17}$$

and so,

$$1 - A_2(1-p)e^{-r_2 N} > 0 \tag{5.18}$$

$$\theta(N) := A_1 p e^{-r_1 N} + A_2(1-p)e^{-r_2 N} > 1. \tag{5.19}$$

If $A_1 p + A_2(1-p) < 1$ then (5.19) does not hold for any $N \geq 0$, thus I assume $A_1 p + A_2(1-p) > 1$. Now,

$$\Phi(1-\alpha) = \omega p N\left(1 - e^{-\beta\Phi}\right) > 0 \text{ for all } N > 0,$$

and the host steady state is given by $f(N) = g(N)$ where

$$\begin{aligned} f(N) &= \frac{1-\alpha}{\beta} \ln\left\{\frac{A_1 p e^{-r_1 N}}{1 - A_2(1-p)e^{-r_2 N}}\right\} \\ g(N) &= \omega p N\left\{1 - \frac{1 - A_2(1-p)e^{-r_2 N}}{A_1 p e^{-r_1 N}}\right\}. \end{aligned}$$

and the pathogen steady state is given by

$$\Phi = \frac{1}{\beta} \ln\left\{\frac{A_1 p e^{-r_1 N}}{1 - A_2(1-p)e^{-r_2 N}}\right\}.$$

Thus $\Phi > 0$

$$\begin{aligned} &\Leftrightarrow \Gamma(N) := \frac{A_1 p e^{-r_1 N}}{1 - A_2(1-p)e^{-r_2 N}} > 1 \\ &\Leftrightarrow \theta(N) > 1 \text{ by (5.18) \& (5.19)} \\ &\Leftrightarrow \theta(N) > \theta(\hat{N}) \text{ where } \hat{N} > 0 \text{ satisfies } \theta(\hat{N}) = 1 \\ &\Leftrightarrow N < \hat{N} \text{ since } \theta \text{ is strictly decreasing.} \end{aligned}$$

Now, $g(0) = g(\hat{N}) = 0$ and $g(N) > 0$ for all $N\epsilon(0, \hat{N})$ by (5.17). Also

$$f(0) = \frac{1-\alpha}{\beta} \ln\left\{\frac{A_1 p}{1 - A_2(1-p)}\right\},$$

and note that $A_2(1-p) < 1$ for $f(0)$ to be defined. Also, $f(\hat{N}) = 0$ with $\hat{N} > 0$ if, and only if,

$$A_1 p + A_2(1-p) > 1$$

and if $A_2(1-p) < 1$ then $f(0) > 0$. Now since $\Gamma(N)$ is a strictly decreasing function $\forall N > \tilde{N}$, (where $\tilde{N} = \frac{\ln(A_2(1-p))}{r_2}$ is the pole of f) it follows that $f(N)$ is a strictly decreasing function $\forall N > \tilde{N}$ and is undefined $\forall N \leq \tilde{N}$.
Thus it suffices to show that $f'(\hat{N}) > g'(\hat{N})$ which is equivalent to

$$\hat{N} > \frac{1-\alpha}{\omega\beta p}.$$

Hence if $A_1 p + A_2(1-p) > 1$ and $\hat{N} > \frac{1-\alpha}{\omega\beta p}$ then there exists a non-trivial steady state $N^*\epsilon(0, \hat{N})$. Moreover, if $A_2(1-p) > 1$ then $N^*\epsilon(\tilde{N}, \hat{N})$. □

When considering the linear stability of these steady states using the Jacobian, we see that the trivial steady state is stable if, and only if,

$$A_1 p + A_2(1-p) < 1.$$

This condition for the stability of the trivial steady state is the same as the no pathogen case. Thus, the hosts can be eradicated if, and only if, their joint average contribution to the next generation is less than unity. However, if $A_1 p + A_2(1-p) > 1$ then it is impossible to completely eradicate the hosts, no matter how large the initial application of pathogen is.
The host-only steady state is stable only if the joint contribution to the next generation exceeds unity and

$$\hat{N} < \frac{1-\alpha}{\omega\beta p}. \tag{5.20}$$

Lemma 5.3.2 also tells us that:

1. For the host-pathogen non-trivial steady state to be biologically realistic the combined average contribution to the next generation must exceed unity and the host only steady state must exceed some critical value. This is consistent with the classical threshold theory for infectious diseases (Kermack & McKendrick 1927) where disease will only increase in a population if the susceptible exceeds a critical level.

2. $N^* < \hat{N}$, that is, the host-pathogen steady state is lower than the host only

steady state. Thus, the presence of the pathogen has had a positive impact on the control of the pest host species.

3. If the virulence of the pathogen is increased (i.e. an increase in ω, α and β) then the non-trivial host steady state, N^*, will decrease. However, if $A_2(1-p) > 1$ then N^* tends to $\tilde{N} = \frac{\ln A_2(1-p)}{r_2} > 0$ and if $A_2(1-p) < 1$ then N^* tends to zero, as the virulence of the pathogen increases. Hence, if resistant hosts develop which are sufficiently fecund then it may be impossible to reduce the host population to a desired level.

4. If the pathogen initially released is not sufficiently virulent, such that (5.20) holds then eventually the pathogen will decay and the hosts will persist. Thus releasing an insufficiently virulent pathogen will in the long run have little effect.

5.3.3 Full Model: Controlled Scheme

I now consider the full model (5.2) with $a > 0$.

Lemma 5.3.3. *There are two steady state solutions. The trivial steady state is given as*

$$(N, \Phi) = \left(0, \frac{a}{1-\alpha}\right) \tag{5.21}$$

which is stable, if and only if,

$$A_1 p e^{-\frac{a\beta}{1-\alpha}} + A_2(1-p) < 1. \tag{5.22}$$

The host-pathogen non-trivial steady state is

$$(N, \Phi) = (N^*, \Phi^*) \tag{5.23}$$

where (N^*, Φ^*) is given by the implicit relation $f(N^*) = g(N^*)$ with

$$f(N^*) = \frac{1-\alpha}{\beta} \ln\left\{\frac{A_1 p e^{-r_1 N^*}}{1 - A_2(1-p)e^{-r_2 N^*}}\right\} - a, \tag{5.24a}$$

$$g(N^*) = \omega p N^* \left\{1 - \frac{1 - A_2(1-p)e^{-r_2 N^*}}{A_1 p e^{-r_1 N^*}}\right\}, \tag{5.24b}$$

$$\Phi^* = \frac{f(N^*) + a}{1-\alpha}. \tag{5.24c}$$

In this case the non-trivial solution is biologically realistic if

$$A_1 p e^{-\frac{a\beta}{1-\alpha}} + A_2(1-p) > 1, \tag{5.25}$$

Proof. Setting $N_{t+1} = N_t = N$ and $\Phi_{t+1} = \Phi_t = \Phi$, the non-trivial steady state

equations are

$$\begin{aligned} 1 &= A_1 p e^{-r_1 N} e^{-\beta\Phi} + A_2(1-p)e^{-r_2 N} \\ \Phi &= \omega p N\left(1 - e^{-\beta\Phi}\right) + \alpha\Phi + a. \end{aligned}$$

Therefore,

$$0 < \frac{1 - A_2(1-p)e^{-r_2 N}}{A_1 p e^{-r_1 N}} = e^{-\beta\Phi} < 1 \tag{5.26}$$

and so,

$$1 - A_2(1-p)e^{-r_2 N} > 0 \tag{5.27}$$

$$\theta(N) := A_1 p e^{-r_1 N} + A_2(1-p)e^{-r_2 N} > 1. \tag{5.28}$$

If $A_1 p + A_2(1-p) < 1$ then (5.28) does not hold for any $N \geq 0$, thus I assume $A_1 p + A_2(1-p) > 1$. Now,

$$\Phi(1-\alpha) - a = \omega p N\left(1 - e^{-\beta\Phi}\right) > 0 \text{ for all } N > 0,$$

and so for $a > 0$, $\Phi > \frac{a}{1-\alpha}$, and the host steady state is given by $f(N) = g(N)$ where

$$\begin{aligned} f(N) &= \frac{1-\alpha}{\beta} \ln\left\{\frac{A_1 p e^{-r_1 N}}{1 - A_2(1-p)e^{-r_2 N}}\right\} - a \\ g(N) &= \omega p N\left\{1 - \frac{1 - A_2(1-p)e^{-r_2 N}}{A_1 p e^{-r_1 N}}\right\}. \end{aligned}$$

and the pathogen steady state is given by

$$\Phi = \frac{1}{\beta} \ln\left\{\frac{A_1 p e^{-r_1 N}}{1 - A_2(1-p)e^{-r_2 N}}\right\}.$$

Thus $\Phi > 0$

$$\begin{aligned} &\Leftrightarrow \Gamma(N) := \frac{A_1 p e^{-r_1 N}}{1 - A_2(1-p)e^{-r_2 N}} > 1 \\ &\Leftrightarrow \theta(N) > 1 \text{ by (5.27) \& (5.28)} \\ &\Leftrightarrow \theta(N) > \theta(\hat{N}) \text{ where } \hat{N} > 0 \text{ satisfies } \theta(\hat{N}) = 1 \\ &\Leftrightarrow N < \hat{N} \text{ since } \theta \text{ is strictly decreasing.} \end{aligned}$$

Now, $g(0) = g(\hat{N}) = 0$ and $g(N) > 0$ for all $N \epsilon (0, \hat{N})$ by (5.26). Also

$$f(0) = \frac{1-\alpha}{\beta} \ln\left\{\frac{A_1 p}{1 - A_2(1-p)}\right\} - a,$$

and note that $A_2(1-p) < 1$ for $f(0)$ to be defined. Also, $f(\bar{N}) = 0$ with $\bar{N} > 0$ if, and

only if,

$$A_1 p e^{-\frac{a\beta}{1-\alpha}} + A_2(1-p) > 1$$

and if $A_2(1-p) < 1$ then $f(0) > 0$. Now since $\Gamma(N)$ is a strictly decreasing function $\forall N > \tilde{N}$, (where $\tilde{N} = \frac{\ln(A_2(1-p))}{r_2}$ is the pole of f) it follows that $f(N)$ is a strictly decreasing function $\forall N > \tilde{N}$ and is undefined $\forall N \leq \tilde{N}$.
Thus it suffices to show that $\bar{N} < \hat{N}$. But, $f(\bar{N}) = 0 > -a = f(\hat{N})$ which is equivalent to $\bar{N} < \hat{N}$ since f is strictly decreasing. Hence if $A_1 p e^{-\frac{a\beta}{1-\alpha}} + A_2(1-p) > 1$ then there exists a non-trivial steady state $N^* \epsilon (0, \hat{N})$. Moreover, if $A_2(1-p) > 1$ then $N^* \epsilon (\tilde{N}, \hat{N})$. □

Hence Lemma 5.3.3 tells us that the hosts will die out if the average contribution to the next generation by the whole host population in the presence of the pathogen is less than unity. Clearly, if $A_1 p + A_2(1-p) < 1$ then (5.22) holds, which means that if the average contribution to the next generation in the absence of the pathogen is less than unity then the host population will die out whether the pathogen is present or not. Also notice that if $A_2(1-p) > 1$ then the host free (trivial) steady state is never stable. Biologically this means that if there are too many resistants being produced with a large average number of offspring, then one cannot eradicate the host population.
Numerical simulations indicate that in some regions of parameter space, the non-trivial steady state is stable (see Figure 5-4). Observe that:

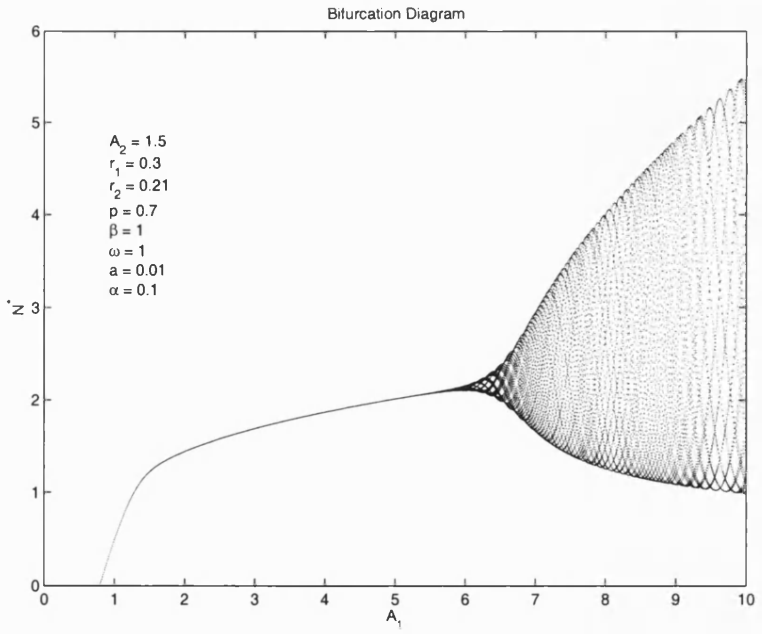


Figure 5-4: Bifurcation diagram for the model system (5.2).

1. If (5.25) holds then $A_1 p + A_2(1-p) > 1$, in other words, if the hosts can persist

under the attack of the pathogen, then they can persist in the absence of the pathogen.

2. If $A_2(1-p) > 1$ then (5.25) holds, that is, if there are sufficiently many resistant hosts produced which are sufficiently fecund then the non-trivial solution is biologically feasible. Moreover, the host population will always persist no matter how fit the susceptible hosts are under the attack of the pathogen, even if large applications of pathogen is applied.

3. If $A_2(1-p) < 1$, then the susceptible hosts must be able to produce (on average) more than one offspring under the attack of the pathogen. Clearly from (5.25), by increasing the application and/or the virulence of the pathogen, one can break this condition.

4. $\Phi^* > \frac{a}{1-\alpha}$, that is the pathogen steady state level in the non-trivial case is larger then that in the trivial case.

5. If (5.25) holds for a given pathogen, i.e. α, β and ω are fixed, then increasing a decreases the host population equilibrium level. If $A_2(1-p) < 1$ then condition (5.25) can be violated and the trivial steady state becomes stable, and so the host population will die out. However, if $A_2(1-p) > 1$ then condition (5.25) can never be broken no matter how large a becomes, and moreover, N^* is bounded below by $\frac{\ln A_2(1-p)}{r_2}$. Biologically this means that if $A_2(1-p) < 1$ then by increasing the applications of pathogen will cause the hosts to die out, and if $A_2(1-p) > 1$ then it is impossible for the hosts to die out no matter how much pathogen is applied.

6. If (5.25) holds then $N^* < \hat{N}$, that is, the non-trivial steady state for the hosts in the presence of the pathogen is lower than that in the absence of the pathogen. Therefore, the addition of the pathogen has had a positive effect on the control of the hosts.

7. $a > 0$ can have a stabilising effect on the model system. In Figure 5-5 we see that for $a = 0$ the non-trivial steady state is unstable, but as a increases the periodic solution is dampened and eventually becomes stable. I have found this to be true for a wide range of parameter values.

5.4 Varying p

In this section we investigate how p affects the steady state levels using analytical and numerical techniques. I first consider the case when no pathogen is present.

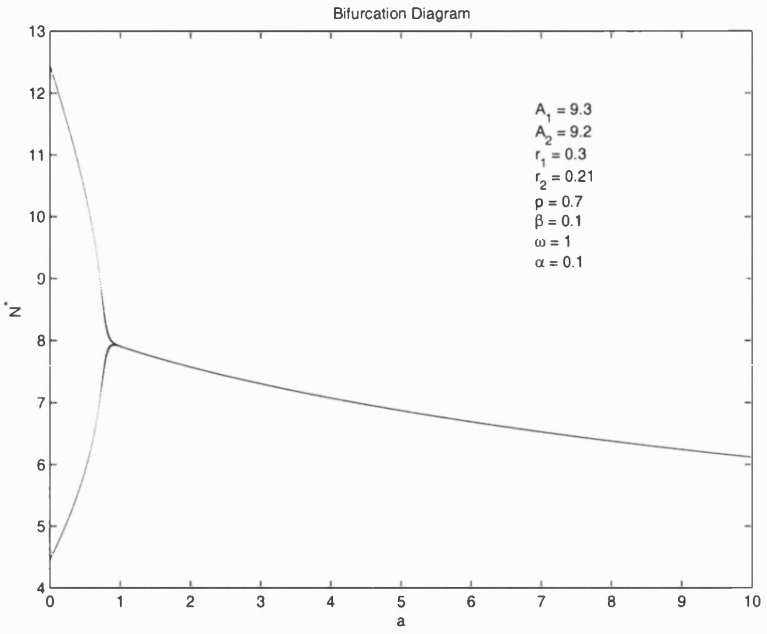


Figure 5-5: Bifurcation diagram for Model (5.2) as a increases.

	$\mathbf{r_1 > r_2}$	$\mathbf{r_1 < r_2}$
$\mathbf{A_1 > A_2}$	$\frac{\partial \hat{N}}{\partial p} > 0$ if $\frac{\ln A_2}{r_2} < \frac{\ln A_1}{r_1}$ $\frac{\partial \hat{N}}{\partial p} < 0$ if $\frac{\ln A_2}{r_2} > \frac{\ln A_1}{r_1}$	$\frac{\partial \hat{N}}{\partial p} > 0$
$\mathbf{A_1 < A_2}$	$\frac{\partial \hat{N}}{\partial p} < 0$	$\frac{\partial \hat{N}}{\partial p} < 0$ if $\frac{\ln A_2}{r_2} > \frac{\ln A_1}{r_1}$ $\frac{\partial \hat{N}}{\partial p} > 0$ if $\frac{\ln A_2}{r_2} < \frac{\ln A_1}{r_1}$

Table 5.3: Necessary and sufficient conditions on the growth parameters in the no pathogen model (5.3) such that the non-trivial steady state is either strictly increasing or decreasing as p increases.

5.4.1 No Pathogen

In Figure 5-6 and Table 5.3 I show how p affects the non-trivial steady state levels depending on the other model parameters when no pathogen is present ($\beta = 0$). These results can be interpreted ecologically in terms of the strength of intraspecific competition acting each subgroup (r_1 and r_2) and their intrinsic growth rates (A_1 and A_2).

In this subsection I assume that $A_1, A_2 > 1$ so that the non-trivial steady state exists for any value of p as predicted in Section 5.3.1.

If the intraspecific competition is lower for the susceptible subclass ($r_1 < r_2$) but they have a larger intrinsic growth rate ($A_1 > A_2$), then $\hat{N}$ is an increasing function of p, that is, the larger the proportion of susceptibles in the population, the greater the

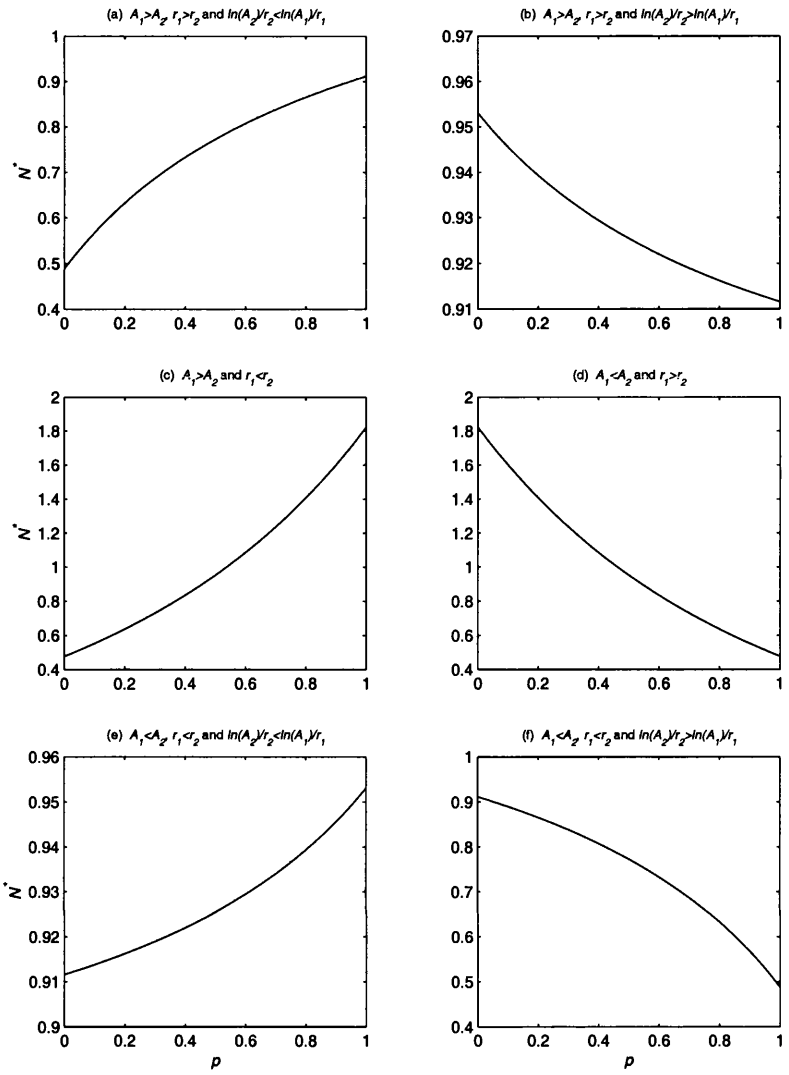


Figure 5-6: How varying p affects the non-trivial steady state in the no pathogen model (5.3). (a) $A_1 > A_2$, $r_1 > r_2$ and $\frac{\ln A_2}{r_2} < \frac{\ln A_1}{r_1}$: Parameter values $A_1 = 1.2$, $A_2 = 1.05$, $r_1 = 0.2$ and $r_2 = 0.1$ (b) $A_1 > A_2$, $r_1 > r_2$ and $\frac{\ln A_2}{r_2} > \frac{\ln A_1}{r_1}$: Parameter values $A_1 = 1.2$, $A_2 = 1.1$, $r_1 = 0.2$ and $r_2 = 0.1$ (c) $A_1 > A_2$ and $r_1 < r_2$: Parameter values $A_1 = 1.2$, $A_2 = 1.1$, $r_1 = 0.1$ and $r_2 = 0.2$ (d) $A_1 < A_2$ and $r_1 > r_2$: Parameter values $A_1 = 1.1$, $A_2 = 1.2$, $r_1 = 0.2$ and $r_2 = 0.1$ (e) $A_1 < A_2$, $r_1 < r_2$ and $\frac{\ln A_2}{r_2} < \frac{\ln A_1}{r_1}$: Parameter values $A_1 = 1.1$, $A_2 = 1.2$, $r_1 = 0.1$ and $r_2 = 0.2$ (f) $A_1 < A_2$, $r_1 < r_2$ and $\frac{\ln A_2}{r_2} > \frac{\ln A_1}{r_1}$: Parameter values $A_1 = 1.05$, $A_2 = 1.2$, $r_1 = 0.1$ and $r_2 = 0.2$

population levels. The converse is true if $A_1 < A_2$ and $r_1 > r_2$, because the resistant subclass has the greatest growth potential.

When there is a trade-off between the benefit of a larger intrinsic growth rate and the cost of greater intraspecific competition ($A_1 > A_2$ and $r_1 > r_2$ or $A_1 < A_2$ and $r_1 < r_2$), whether the population is an increasing or decreasing function of p depends on

the steady state levels in the absence of an intraspecific competitor. In the case where $A_1 > A_2$ and $r_1 > r_2$ then the steady state increases with p if the steady state in the absence of the resistant subclass is higher than that in the absence of the susceptible subclass ($\frac{\ln A_1}{r_1} > \frac{\ln A_2}{r_2}$). Otherwise the steady state decreases with p. The converse is true if $A_1 < A_2$ and $r_1 < r_2$.

As we have seen in Chapter 4, it is often assumed that there is a trade-off between susceptibility to disease and differences in birth rate (A) and/or resistance to crowding (r) (Bowers et al. 1994). This is because a benefit gained in one area of a species life history will trade-off with a cost in another. However, in the absence of the pathogen we are more likely to see the that the susceptible subclass will have greater growth potential than the resistants, since the trade-off in susceptibility is of no importance. Hence, one would expect that $\frac{\ln A_1}{r_1} > \frac{\ln A_2}{r_2}$, however, it is still possible for there to be a trade-off in birth rate and resistance to crowding. Moreover, as Bowers et al. (1994) discusses, resistance is most likely to evolve in species with high intrinsic birth rates and low resistance to crowding. This is because of the opportunities they present for resistant mutants appearing and then growing rapidly in proportion within the population.

5.4.2 The Complete System

To explore the effect of p on the host population levels I use numerical simulations (since the algebraic relations which we need to explore are intractable). In Figures 5-7 to 5-9 I present some typical results and I use these to highlight how the host-pathogen interaction interacts with the parameter p. The parameters chosen for these numerical simulations were arbitrary but were chosen to demonstrate the range of outcomes which have been observed in extensive numerical simulations.

In Figure 5-7 I observe three distinct cases.

Case 1: Host levels increase with p. See Figure 5-7 (c). The cost of resistance, in terms of intraspecific competition is large and so increasing the proportion of susceptibles in the population results in an increase in total host levels. Pathogen levels increase with p as the pathogen induced mortality increases with the hosts increase.

Case 2: Host levels decrease with p. See Figure 5-7 (b),(d),(e) & (f). This arises when the relative effect of pathogenicity on the susceptible hosts is large, so that an increase in the proportion of susceptible hosts results in more pathogen induced mortality. For the parameters used in these figures, pathogen levels initially increase despite a decrease in total host numbers because there is an increase in the proportion of hosts which are susceptible. When host numbers fall sufficiently, this causes a reduction in pathogen numbers. In Figure 5-7 (e)

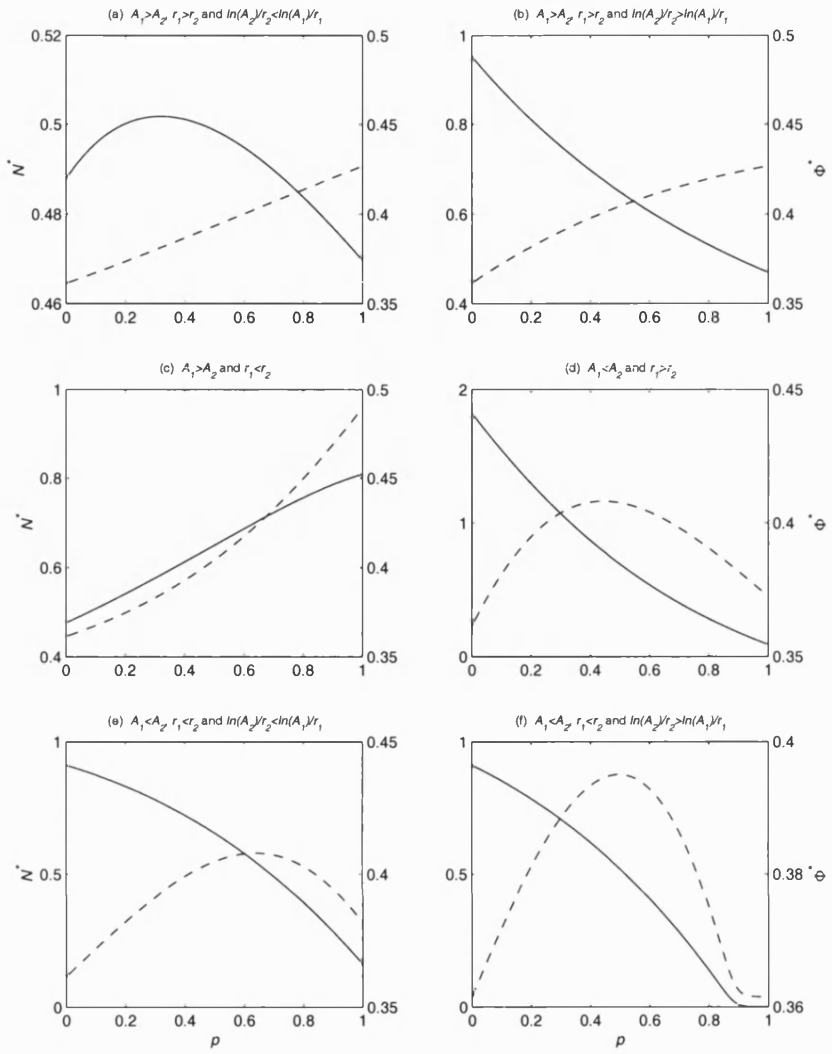


Figure 5-7: How varying p affects the steady states in the full model system (5.2). The solid and dashed lines represent the host and pathogen steady state levels respectively. The host growth parameters are as in Figure 5-6 with $a = 0.1338$, $\beta = 0.2071$, $\alpha = 0.6299$ and $\omega = 0.6072$. For the parameter values used here, $\hat{N} > \frac{1-\alpha}{\omega\beta p}$ for all values of $p\epsilon(0, 1]$. Thus the host steady state levels are the same for the $\beta = 0$ and the $\beta > 0, a = 0$ cases.

host levels decrease as p increases, where they previously increased when no pathogen was present (see Figure 5-6 (e)). This is because the pathogen benefits from an increase in the host susceptibility and therefore increases. This results in a decline of the hosts from pathogen induced mortality. In Figure 5-7 (f) the host levels decrease as in Figure 5-6 (e). However, in this case the hosts vanish as the proportion of susceptibles increases. This is not true in the case when there is no external input ($a = 0$).

Case 3: Host maximum for $p\epsilon(0,1)$. See Figure 5-7 (a). The initial increase in host levels as p increases reflects the larger intrinsic growth rate of the susceptible population compared with the resistant group. Increased host levels results in increased pathogen levels which eventually causes a decline in the host population levels (since a greater proportion of hosts are susceptible to disease induced mortality). Pathogen levels increase throughout the domain due to a combination of external input (a) and pathogen induced host mortality.

From the analysis of the steady states of the hosts in the external ($a > 0$) and no external ($a = 0$) input cases, it follows that increasing the amount of external input (increasing a) decreases the non-trivial host steady state levels for all positive values of p. In particular, the non-trivial host steady state in the external input case must be less than the no external input case for all positive values of p. Thus any amount of external input is beneficial over no external input. This is demonstrated in Figure 5-8. Moreover, for p sufficiently small, in cases (a),(b),(c),(e) and (f) the host only steady state, $\hat{N}$, is stable when there is no external input since $\hat{N} < \frac{1-\alpha}{\omega\beta p}$. When p is sufficiently large the host only steady state loses stability and the host-pathogen non-trivial steady state, N^*, becomes stable. Biologically this means that if the host population has sufficiently many resistant hosts then the natural epidemics of the pathogen will have no effect. In case (d), $\hat{N}$ is stable for all values of p, thus the natural epidemics have no effect on the host population.
One may also observe population outbreaks for some parameter sets. In the absence of pathogen interactions (see Figure 5-9 (a)), the host population levels are stable. Introducing the pathogen (see Figure 5-9 (b)) destabilises the system such that, if p is sufficiently large, the dynamics are driven unstable with population outbreaks larger than that in the case where no pathogen is present. However, when an external input is included the population outbreaks are reduced in magnitude and outbreaks occur for a smaller range of p. I suggest that this phenomenon is due to the inhibiting effects of the external input on the growth of the hosts.

5.5 Conclusions & Discussion

I have analysed the population dynamics of host and pathogen as a prelude to a study of the management of resistance and the effects of an external input of pathogen.
I have shown that if the hosts average contribution to the next generation is less than unity (whether the pathogen is present or not) then they will die out. However, if the contribution is greater than unity then under a scheme with no external input of pathogen the hosts will always be able to persist, whilst under an external input scheme, this may not be the case. This suggests that if the pathogen is insufficiently virulent then a single application of pathogen and relying on the resultant epidemic may not

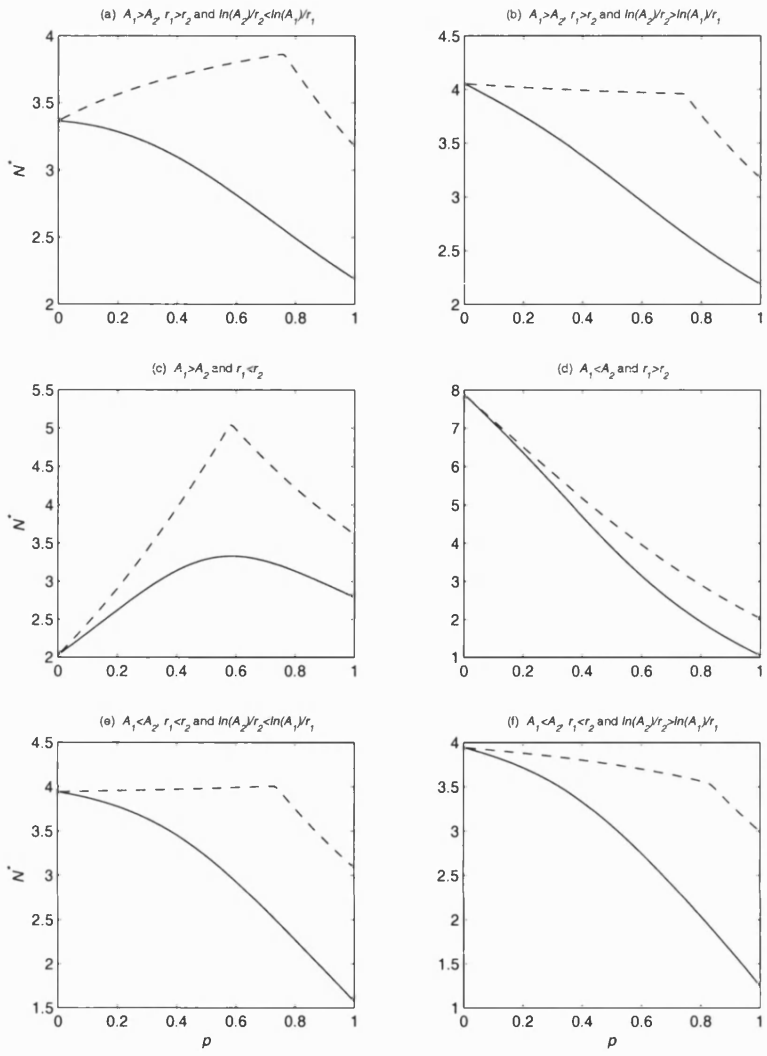


Figure 5-8: How varying p affects the host steady state in the no external input ($a = 0$) and external input ($a > 0$) cases. In all the figures ((a) to (f)), the dashed line represents the host steady state when $a = 0$ and the solid line represents the host steady state when $a = 0.1388 > 0$. The pathogen parameters are $\omega = 0.6072$, $\beta = 0.2071$ and $\alpha = 0.6299$. The host parameters are (a) $A_1 = 2.2$, $A_2 = 1.4$, $r_1 = 0.2$ and $r_2 = 0.1$ (b) $A_1 = 2.2$, $A_2 = 1.5$, $r_1 = 0.2$ and $r_2 = 0.1$ (c) $A_1 = 2.2$, $A_2 = 1.5$, $r_1 = 0.1$ and $r_2 = 0.2$ (d) $A_1 = 1.5$, $A_2 = 2.2$, $r_1 = 0.2$ and $r_2 = 0.1$ (e) $A_1 = 1.5$, $A_2 = 2.2$, $r_1 = 0.1$ and $r_2 = 0.2$ (f) $A_1 = 1.4$, $A_2 = 2.2$, $r_1 = 0.1$ and $r_2 = 0.2$. The sharp change in behaviour of the dashed line corresponds to a change in stability of the steady states. Closer inspection of the plots reveals that the change in hosts steady state is more smooth than it appears in the plots.

have the desired level of host control. However, by including an external input, the pathogen will always have a positive impact on host control. Moreover, the external input may reduce the level of host population outbreaks and reduce the parameter

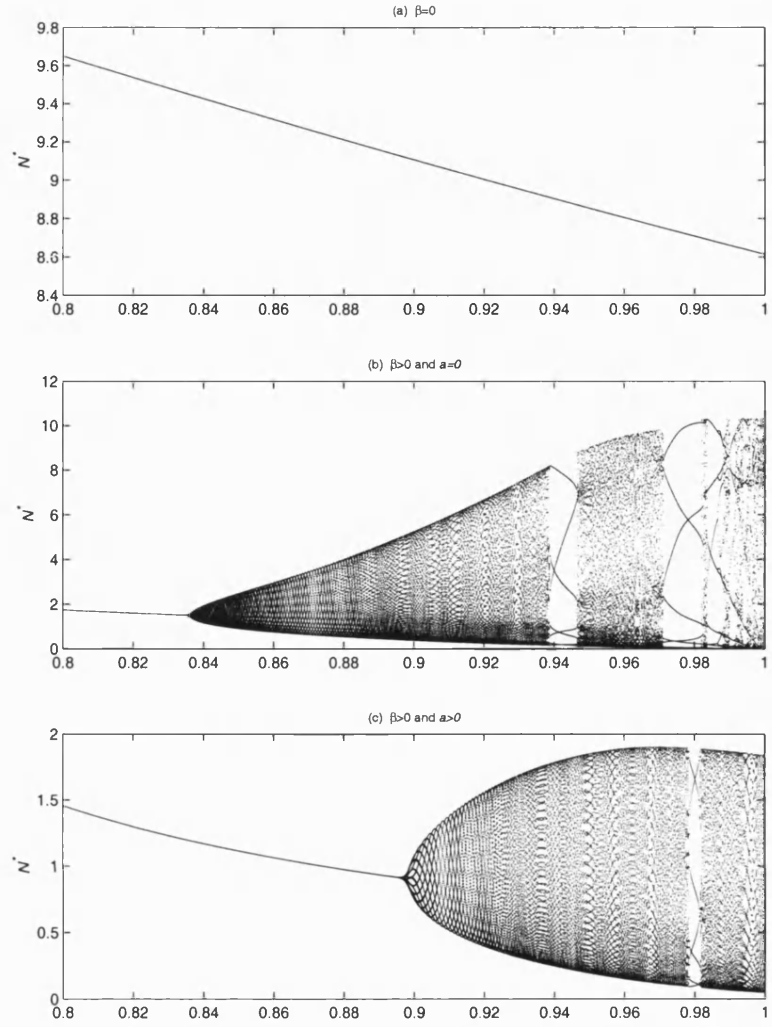


Figure 5-9: How p affects the stability of the hosts steady state. The parameter values are $A_1 = 5.6$, $A_2 = 4.59$, $r_1 = 0.2$, $r_2 = 0.1$, $a = 0.3$, $\beta = 1.78$, $\omega = 1.07$ and $\alpha = 0.06$.

range for which an outbreak may occur.

In both the external and no external input schemes, the level of control over the hosts may depend on the fitness of the resistant hosts. If the resistant hosts are *fit* (i.e. $A_2(1-p) > 1$) then desired levels of control over the hosts may not be possible. Moreover, applying more pathogen, or increasing pathogenicity, may have little effect on the host population levels.

In this chapter I have shown that the role of p can have a large impact on the control of the hosts. If large proportions of resistant hosts develop then control may be impossible. Throughout this chapter I have assumed that p is constant in order to fully understand the impact of the externally controlled fungal pathogen has on the system. However, it may be more realistic to assume that this probability is dependent on the abundance of

the free-living pathogen. Empirically one would expect that as the abundance of free-living pathogen increased, the probability of remaining susceptible at each generation will decrease. Thus one might expect that p is a decreasing function of Φ_t. However, this scenario is not considered in this thesis, but is to be considered in future work.

This chapter is focussed entirely on the temporal interactions of an insect host and its pathogen. The majority of these interactions take place in a spatial context and important behaviours such as patchiness of insect distributions and refugia positioning, can be modelled in a spatially extended system which is the topic of the next chapter.

It is well understood that for a polymorphism to exist between susceptible and resistant hosts that some degree of cost must be associated with resistance (see Gillespie (1975)). Usually the cost comes in the form of reduced fecundity or lack of competitive ability. These costs are often regarded as trade-offs between the susceptible and resistant traits, and moreover, it is thought that polymorphism is least likely to occur when the traits of two strains are similar (see Antonovics & Thrall (1994) and Bowers et al. (1994)). Recent studies have shown that such costs are not always as easily attributed. Ferrari, Muller, Kraaijeveld & Godfray (2001) suggested that there was no correlation in defensive ability to a fungal pathogen and fecundity in pea aphids. More subtle costs have been investigated, such as host plant specialisation in pea aphids (Ferrari & Godfray 2003). Other costs may include the correlation between host plant quality and competition between biotypes of pest. This suggests that new investigations into tritrophic insect-pathogen-plant system may lead to deeper insight into the speciation and development of resistance in insects.

Clearly the models presented in this chapter have assumed that p is constant. Rather than using this model formulation, one could formulate the problem in terms of evolutionary adaptive dynamics where there exists a trade-off between susceptibility and some other model parameter (such as A) using the full model (5.2) with $p = 1$. Then following Metz et al. (1992) one can calculate the time-averaged logarithmic growth rate to determine the ESS if it exists, and whether the ESS is convergent stable. This idea is similar to that dicussed in Chapter 4 for the continuous-time model. Thus conditions can be found (if they exist) to get branching and hence a dimorphic population, where one strain is more susceptible to disease than the other.

Chapter 6

Coupled Map Lattices

6.1 Motivation and Background

6.1.1 Chapter Outline

In this chapter I consider a spatial extension to the discrete-time model that is considered in Chapter 5. To do this I use coupled map lattices. In order to analyse the coupled map lattice (CML), I first consider a general model. To analyse this general model I make a link between the CML and integro-difference equations. Then I derive general conditions for dispersal-driven instabilities on the CML to determine under what conditions spatial patterning can be observed.
I apply this theory to the discrete-time model presented in Chapter 5. I then investigate the case where the local temporal dynamics are unstable, but can give rise to spatially structured populations on the CML.

6.1.2 Motivation

Although the population model in Chapter 5 considers the temporal dynamics of a host-pathogen system, it does not take into account the spatial dynamics. In Holland, Perry & Winder (1999), Winder, Alexander, Holland, Woolley & Perry (2001) and Holland, Winder & Perry (2000) the authors have found spatial patterning in insect species. In particular, in Winder et al. (2001) the authors demonstrated that there exists spatial patterning in the predator-prey dynamics between two species of aphid and a generalist beetle predator. Moreover, the authors observe a positive, lagged beetle response to the aphids spatial pattern; and conversely, the aphids displayed a negative, lagged response to beetle spatial pattern. In Holland et al. (2000) the spatial distributions of beneficial arthropods (beneficial arthropods generally refer to terrestrial arthropods, mostly insects, that are important in the biological control of agronomic pests, typically through predation or parasitism) before and after the application of dimethoate (an insecticide used to kill mites and insects systematically and on contact) were observed.

The authors where able to show that before spraying, these non-target insects showed spatial patterning. After spraying the spatial patterning significantly altered, with some species not showing much patterning at all.
In this chapter I investigate some of the possible mechanisms for this spatial patterning using CML's.

6.1.3 Background Reading

The CML depicts an environment which is divided into local habitats that contain resources (i.e. plants) for herbivorous insects with discrete generations. The emerging adult insects either remain at their present site and oviposit or move to a neighbouring site and oviposit. The remainder of the life-cycle is spent at the current site.
This environment is generally represented mathematically by a two-dimensional square grid of lattice points (cells/sites/patches), with each lattice point representing a habitat for a local population. Most single species CML formulations assume that within each generation there are two distinct phases:

The dispersal phase: In this phase a certain fraction of hosts, μ, leave the currently occupied lattice point from which they emerged to a neighbouring lattice point. A fraction $1-\mu$ remain in their current lattice point. Thus the equation of the dispersal stage is

$$N'_{i,t} = (1-\mu)N_{i,t} + \frac{\mu}{m}\sum_{j\in\mathcal{N}} N_{j,t} \tag{6.1}$$

where $N_{i,t}$ and $N'_{i,t}$ denote the pre-dispersal and post-dispersal host densities at lattice point i at generation t respectively, $\mathcal{N}$ is the interacting neighbourhood and m is the number of lattice points in the neighbourhood. I assume that the interacting neighbourhood is the eight nearest neighbours in the two dimensional lattice, however using the theory developed in this chapter more complex and indeed perhaps more realistic neighbourhoods can be investigated with little difficulty.

Within patch phase: In this phase the local population reproduces and matures within its patch. It is assumed that all patches are identical in quality. Mathematically we have

$$N_{i,t+1} = f(N'_{i,t}) \tag{6.2}$$

where f represents the growth rate for the population (see Chapter 5 for examples).

There has been some discussion into the formulation of the single species CML. In Bascompte & Solé (1994) the authors assumed that population growth and dispersion

act simultaneously. The model that they considered was

$$N_{t+1}(i,j) = f(N_t(i,j)) + D\nabla^2 N_t(r)$$

where $N_t(i,j)$ is the density of hosts at generation t at the $(i,j)^{th}$ lattice position, D is the dispersal rate, and

$$\nabla^2 N_t(r) = N_t(i-1,j) + N_t(i+1,j) + N_t(i,j-1) + N_t(i,j+1) - 4N_t(i,j).$$

However, as discussed in Hassell, Miramontes, Rohani & May (1995) and Bascompte & Solé (1995) there are certain problems with this formulation. Hassell et al. (1995) argue that the result 'as the dispersal rate increases the dynamics become increasingly unstable', is surprising for single species models. Hassell et al. (1995) conclude that this counter-intuitive result arises solely from the biologically unreasonable assumption that individuals may fail to survive and yet may still disperse, leading to negative population densities at some patches. Therefore, Hassell et al. (1995) argue that the growth and dispersal must be separated in the CML formulation. To that end, Hassell et al. (1995) split the host class into two age classes; one for adults and one for larvae. From this structure the authors show that the addition of dispersal has no effect on the stability of the homogeneous steady state. However, in order to achieve this, the authors had to use a specific form of population model.
On a technical note, the neighbourhood may differ from patches on the boundary of the arena. Generally three scenarios are considered:

Periodic Periodic (or cyclic) boundaries have opposite edges of the arena joining together. Thus if the arena is a two-dimensional lattice, then using periodic boundary conditions effectively turns the arena into a torus. This is clearly unrealistic, but has the advantage that all lattice points are equivalent, as there are no edge effects.

Absorbing When an individual disperses across the boundary, it is assumed to be lost.

Reflective Individuals are not allowed to cross the boundary, and so are reflected back into the arena.

Equations (6.1) and (6.2) along with prescribed boundary conditions define the CML. Further reading on CML's can be found in Tilman & Kareiva (1997), Kaneko (1992) and Hassell (2000).
One key question has been asked of CML's; 'under what conditions can dispersion destabilise population dynamics, or does the dispersal have a stabilising effect'? This idea is discrete-time equivalent to Turing instabilities (Murray 1989) for reaction diffusion

systems. Many authors have attempted this question (Rohani, May & Hassell 1996, Rohani & Ruxton 1999a, Rohani & Ruxton 1999b, Neubert, Caswell & Murray 2002, Bascompte & Solé 1994) but they only cover stability for specific types of models as opposed to a general framework. In this chapter I draw up this general framework by using the analysis for integro-difference equations.

Integro-difference equations are simple models that have discrete-time population dynamics and a continuous spatial habitat. The concept of the model is much the same as the CML in that there are two distinct stages; a growth stage and a redistribution stage. The composition of these two stages yields the integro-difference equation

$$N_{t+1}(x) = \int_{\Omega} k(x,y) f(N_t(y)) \mathrm{d}y \tag{6.3}$$

where $N_t(x)$ is the population at generation t at point x in space, $k(x,y)$ is the redistribution kernel where $k(x,y)dy$ is the probability that an individual, N, at x originated from an interval of length dy about y, and Ω is the spatial domain. It is often the case that the redistribution kernel depends on the absolute location or on the relative distance, in which case we may rewrite (6.3) as

$$N_{t+1}(x) = \int_{\Omega} k(x-y) f(N_t(y)) dy. \tag{6.4}$$

Different types of redistribution kernels can be used in order to best fit the species that is being model. An excellent review of integro-difference equations and their analysis can be found in Neubert, Kot & Lewis (1995).

For two species CML models, one model in particular has drawn some interest; the spatially extended Nicholson-Bailey model that features in Comins, Hassel & May (1992). The authors show that a wide range of spatially structured patterns are observed which depend on the fractions of hosts and parasitoids that disperse in each generation. These spatial patterns arise from the locally unstable population dynamics, as opposed to dispersal-driven instabilities. However, due to the unstable nature of this model, the authors have to make some numerical adjustments to compensate for this. In this chapter I show that these types of spatial behaviours are possible for a more realistic population model.

6.2 Model & Analysis

Throughout this chapter I assume that the CML has periodic boundary conditions so that each lattice point is equivalent. However, many of the results and observations presented here can be seen with other boundary conditions.

Let us first consider the case where a single species can disperse over the CML.

6.2.1 Single Species Interactions

Rather than splitting the host population as considered in Hassell et al. (1995), I suggest the following formulation:

$$N_{i,t+1} = (1-\mu)f(N_{i,t}) + \frac{\mu}{m}\sum_{j\in\mathcal{N}} f(N_{j,t}) \quad \text{for} \quad i \in \mathcal{L} \tag{6.5}$$

where $\mathcal{L}$ is the set of all lattice points. In this formulation the density dependent growth acts at each site before dispersal, therefore avoiding dead individuals dispersing. This two stage process is similar to that exhibited by integro-difference equations (see Section 6.2.2).
In the following lemma I will show that dispersal driven instabilities are not possible with single species models.

Lemma 6.2.1. *For a single species CML* (6.5) *with cyclic (periodic) boundary conditions, dispersion cannot destabilise the spatially homogeneous equilibrium.*

Proof. Suppose that the local population dynamics given by $N_{t+1} = f(N_t)$, has a unique positive equilibrium that is linearly stable, that is, there exists a unique $N^* > 0$ such that $N^* = f(N^*)$ where $|f'(N^*)| < 1$.
Now suppose we spatially extend the local population using the CML (6.5) which has periodic boundary conditions. Suppose that the lattice has $n \times n$ lattice points which I label in the following way:

$$\begin{array}{llll} 1 & 2 & \dots & n \\ n+1 & n+2 & \dots & 2n \\ \vdots & \vdots & & \vdots \\ n^2-n+1 & n^2-n+2 & \dots & n^2 \end{array} \tag{6.6}$$

Then (6.5) can be written as

$$\mathbf{U}_{t+1} = \mathbf{K}\mathbf{F}(\mathbf{U}_t) \tag{6.7}$$

where $U_i = N_i$ for $i = 1 \dots n^2$, $F_i(U_i) = f(N_i)$ for $i = 1 \dots n^2$ and $\mathbf{K} \in \mathbb{R}^{n^2 \times n^2}$ is the redistribution matrix whose rows and columns are made from the diffusion coefficient. Hence the rows and columns of $\mathbf{K}$ must sum to unity since redistribution must conserve mass as the boundary conditions are periodic. The exact nature of $\mathbf{K}$ depends on the local dispersion rules, however the matrix has a block structure. An example of this matrix can be found in Figure 6-3.
Now if there is no diffusion then $\mathbf{K} = \mathbf{I}$, the identity matrix, and hence (6.7) becomes

$$\mathbf{U}_{t+1} = \mathbf{F}(\mathbf{U}_t)$$

and hence the homogeneous state state is given by

$$\mathbf{U}^* = \mathbf{F}(\mathbf{U}^*) \quad \text{i.e.} \quad N^* = f(N^*).$$

Now linearising about this homogeneous steady state, $\mathbf{U}^*$, gives

$$\mathbf{V}_{t+1} = \mathbf{KJV}_t$$

for the growth rate of the spatially heterogeneous perturbation $\mathbf{V}_t$, where

$$\mathbf{J} = \text{diag}\left\{f'(N^*), \ldots, f'(N^*)\right\} \in \mathbb{R}^{n^2 \times n^2}.$$

It follows that if $\rho(\mathbf{KJ}) < 1$ (where $\rho(\cdot)$ is the eigenvalue of the matrix with greatest modulus) then $\mathbf{U}^*$ is stable to heterogeneous perturbations, i.e. dispersion cannot destabilise.
Now

$$\begin{aligned}
\rho(\mathbf{KJ}) &\leq \|\mathbf{KJ}\|_2 \\
&\leq \|\mathbf{K}\|_2 \|\mathbf{J}\|_2 \\
&\leq \sqrt{\|\mathbf{K}\|_1 \|\mathbf{K}\|_\infty} \|\mathbf{J}\|_2 \\
&= \|\mathbf{J}\|_2 \text{ since } \|\mathbf{K}\|_1 = \|\mathbf{K}\|_\infty = 1 \\
&= \rho(\mathbf{J}) \text{ since } \mathbf{J} = \mathbf{J}^T \\
&< 1
\end{aligned}$$

□

What Lemma 6.2.1 is saying is that if the local population dynamics are stable, then the addition of dispersion will have no destabilising effect. Moreover, this result is independent of the grid size, and the neighbourhood of dispersion, and of the fractions of hosts dispersing, which is consistent with the formulation in Hassell et al. (1995). Therefore, the CML (6.5) produces biologically and mathematically realistic results, which does not rely on splitting the host population into two age classes.
This result can be seen numerically in Figure 6-1. For this figure I use the Ricker model, and for the parameters used there exists a unique positive stable steady state. Thus Lemma 6.2.1 predicts that the homogeneous steady state will be stable since the local dynamics are stable. As we see in the figure, as time evolves the initial random perturbations from the homogeneous steady state decay and the CML settles down to the homogeneous steady state.
Figure 6-2 (a) shows that the average host density per patch tends to the homogeneous steady state monotonically. This behaviour is consistent with that of the non-spatial

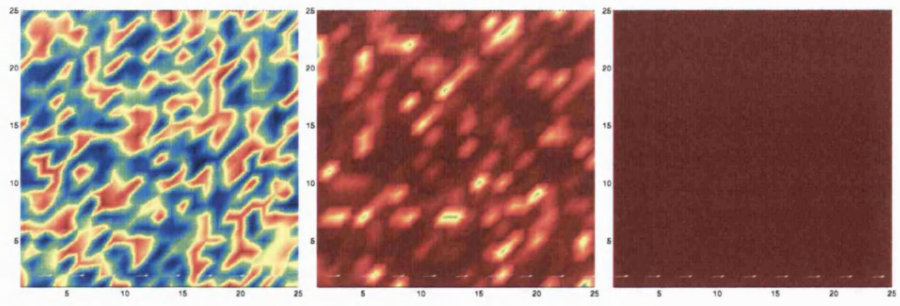


Figure 6-1: A single species CML with periodic boundary conditions. The local dynamics are given by the Ricker model $N_{t+1} = AN_t e^{-rN_t}$. The model parameters are $\mu = 0.1$, $A = 2.3$ and $r = 1$. The hosts disperse to their eight nearest neighbours with equal weighting. The grid size is 25. Areas on the grid in red are high densities and areas in blue are low densities. The left hand figure is the initial random densities. The centre figure is the CML after 4 generations. The right hand figure is the CML after 10 generations.

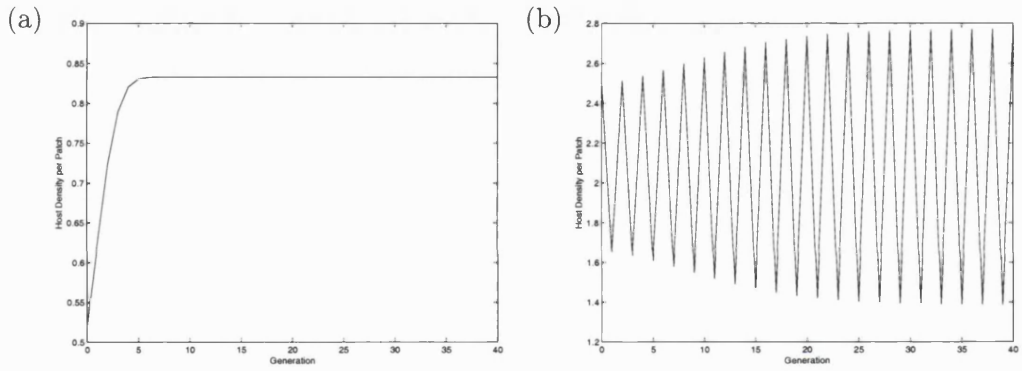


Figure 6-2: Host density per patch. In the above figures the average host density per patch is plotted against time. In (a) the model parameters are as in Figure 6-1. In (b) the model parameters are as in (a) but with $A = 8$.

model. In Figure 6-2 (b) the value of A has been increased to 8 so that the non-spatial model has an unstable positive steady state that oscillates with period two. This is clearly mimicked by the CML where after a transient period the CML switches between two homogeneous population levels in subsequent generations.

As a side note, by representing the CML as (6.7), once the dispersion matrix $\mathbf{K}$ has been prescribed, the numerical simulations for the CML are straightforward. This is because future populations can easily be calculated by matrix multiplication. The structure of the dispersion matrix $\mathbf{K}$ is shown in Figure 6-3 for a 2D single species model where hosts disperse to there eight nearest neighbours. Notice that the matrix is obviously symmetric and the rows and columns sum to unity, since the dispersion rules are symmetric and the boundary conditions are periodic.

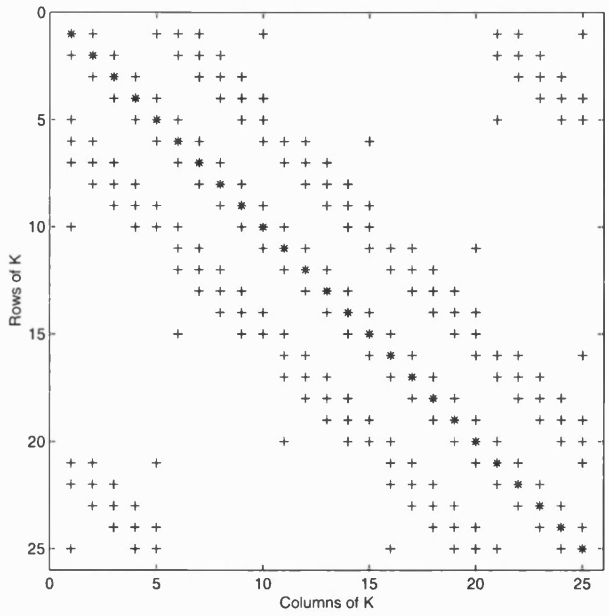


Figure 6-3: The structure of the dispersion matrix $\mathbf{K}$ for a 2D single species model where hosts disperse to their eight nearest neighbours. The boundary conditions are periodic. The $*$'s denote elements of $\mathbf{K}$ whose value is $1-\mu$, where μ is the dispersion coefficient. The $+$'s denote elements of $\mathbf{K}$ whose value is $\mu/8$. The lattice has 5×5 lattice points.

I now extend the single species CML to two interacting species.

6.2.2 Two Interacting Species

Now suppose that we wish to spatially extend the two interacting species model

$$N_{t+1} = f(N_t, P_t) \tag{6.8a}$$
$$P_{t+1} = g(N_t, P_t). \tag{6.8b}$$

Then extending (6.5) to two species we have

$$N_{i,t+1} = (1-\mu_N) f(N_{i,t}, P_{i,t}) + \frac{\mu_N}{m} \sum_{j \in \mathcal{N}} f(N_{j,t}, P_{j,t}) \tag{6.9a}$$
$$P_{i,t+1} = (1-\mu_P) g(N_{i,t}, P_{i,t}) + \frac{\mu_P}{m} \sum_{j \in \mathcal{N}} g(N_{j,t}, P_{j,t}). \tag{6.9b}$$

In this formulation I assume that both species have the same dispersion rules but may have differing dispersion coefficients μ_N and μ_P. However, the following results can be extended to differing dispersion rules as long as each rule assumes that mass is conserved, i.e. there is no mortality during dispersal.

Now, one important question to ask is 'can dispersal destabilise locally stable population dynamics?' These types of questions have been posed for reaction-diffusion equations which was first discussed in Turing's seminal paper on the chemical basis of morphogenesis (Turing 1952). This theory has led to explanations for mammalian coat patterns, butterfly wing patterns and hair patterns to name but a few.
To answer such a question for the CML I first develop the notion in one spatial dimension (so that the CML is a line of coupled lattice points that are joined at the ends due to the periodic boundary conditions to form a ring), I then extend this notion to two spatial dimensions.

The 1D CML

The key to the following analysis is to notice the structure of (6.9). By noticing that the growth terms act before dispersion, it is possible to formulate the one-dimensional CML in terms of an integro-difference model by choosing a special redistribution kernel. The one spatial dimensional CML of n lattice points is

$$N_{i,t+1} = (1-\mu_N)f(N_{i,t},P_{i,t}) + \frac{\mu_N}{2}\left(f(N_{i+1,t},P_{i+1,t}) + f(N_{i-1,t},P_{i-1,t})\right) \tag{6.10a}$$

$$P_{i,t+1} = (1-\mu_P)g(N_{i,t},P_{i,t}) + \frac{\mu_P}{2}\left(g(N_{i+1,t},P_{i+1,t}) + g(N_{i-1,t},P_{i-1,t})\right) \tag{6.10b}$$

for $i = \{1,2,\ldots,n\}$. This is equivalent to the one dimensional integro-difference model

$$N_{t+1}(x) = \int_\Omega k_N(x-y)f(N_t(y),P_t(y))\mathrm{d}y \tag{6.11a}$$

$$P_{t+1}(x) = \int_\Omega k_P(x-y)g(N_t(y),P_t(y))\mathrm{d}y \tag{6.11b}$$

where $x \in [0,1] = \Omega$ and the redistribution kernels are given by

$$k_j(x-y) = \left[(1-\mu_j)\delta(x-y) + \frac{\mu_j}{2}\left\{\delta(x-y+1/n) + \delta(x-y-1/n)\right\}\right] I(|x-y|) \tag{6.11c}$$

for $j \in \{N,P\}$, where $\delta(\cdot)$ is the usual Dirac delta function, and

$$I(|x-y|) = \begin{cases} 1 & \text{if } |x-y| \in \mathcal{L} \\ 0 & \text{otherwise} \end{cases} \tag{6.11d}$$

is the indicator function, and $\mathcal{L} = \{0, 1/n, 2/n, \ldots, 1\}$ is the set of lattice points.
Clearly (6.10) is equivalent to (6.11) since the integro-difference model has a two stage process. Firstly the growth phase acts, followed by dispersal. Although the integro-difference model acts over all space (since the space is a continuum) only points on the lattice are picked up and moved due to the indicator function, I, and move to adjacent

lattice points. All other points in the continuum do not move. Moreover,

$$\begin{aligned}\int_\Omega k_j(x)\mathrm{d}x &= (1-\mu_j)\int_\Omega \delta(x)I(|x|)\mathrm{d}x + \frac{\mu_j}{2}\int_\Omega \delta(x+1/n)I(|x|)\mathrm{d}x \\ &\quad + \frac{\mu_j}{2}\int_\Omega \delta(x-1/n)I(|x|)\mathrm{d}x \\ &= 1-\mu_j+\frac{\mu_j}{2}+\frac{\mu_j}{2} \\ &= 1\end{aligned}$$

for $j \in \{N, P\}$. Hence there is no mortality during dispersal.
Since (6.10) is equivalent to (6.11) we may use theory developed for integro-difference for the CML. Therefore, I now give conditions for dispersion-driven instabilities using theory developed in Neubert et al. (1995).

Lemma 6.2.2. *For the two interacting species model* (6.8)*, the spatially extended one dimensional CML* (6.10) *(or equivalently* (6.11)*) can be destabilised by dispersion if, and only if, one or more of the following inequalities are violated:*

$$1 - \mathrm{tr}(\mathbf{KJ}) + \det(\mathbf{KJ}) > 0 \tag{6.12a}$$

$$1 + \mathrm{tr}(\mathbf{KJ}) + \det(\mathbf{KJ}) > 0 \tag{6.12b}$$

where $\mathrm{tr}(\cdot)$ *and* $\det(\cdot)$ *are the trace and determinant of the matrix respectively, and*

$$\mathbf{K} = \begin{bmatrix} 1-\mu_N+\mu_N\cos\left(\frac{\omega}{n}\right) & 0 \\ 0 & 1-\mu_P+\mu_P\cos\left(\frac{\omega}{n}\right) \end{bmatrix}$$

where n *is the number of lattice points in the CML and* ω *is the wave number, and* $\mathbf{J}$ *is the Jacobian of* (6.8).

Proof. Consider a linearly stable unique positive steady state, $N^*, P^* > 0$, of (6.8) where

$$\begin{aligned}N^* &= f(N^*, P^*) \\ P^* &= g(N^*, P^*).\end{aligned}$$

Then assuming periodic boundary conditions, then the steady state (N^*, P^*) is a spatially uniform (homogeneous) steady state of the integro-difference system (6.11) (and equivalently the CML (6.10)). Now consider perturbations from the homogeneous steady state of the form

$$\begin{aligned}N_t(x) &= N^* + n_t(x) \\ P_t(x) &= P^* + p_t(x).\end{aligned}$$

For sufficiently small perturbations, n_t and p_t, we may linearise about the steady state (N^*, P^*) to get

$$n_{t+1}(x) = \int_\Omega k_N(x-y)\left[J_{11}n_t(y) + J_{12}p_t(y)\right]\mathrm{d}y \tag{6.13a}$$

$$p_{t+1}(x) = \int_\Omega k_P(x-y)\left[J_{21}n_t(y) + J_{22}p_t(y)\right]\mathrm{d}y \tag{6.13b}$$

since $\int_\Omega k_j(x)\mathrm{d}x = 1$, and where J_{ij} are the elements of the Jacobian, $\mathbf{J}$.
Now consider the Fourier transform pair

$$\hat{f}(\omega) = \int_{-\infty}^{+\infty} e^{i\omega x} f(x)\mathrm{d}x$$

$$f(\omega) = \frac{1}{2\pi}\int_{-\infty}^{+\infty} e^{-i\omega x}\hat{f}(\omega)\mathrm{d}\omega$$

and the transformed perturbations

$$\hat{n}_t(\omega) = \int_{-\infty}^{+\infty} e^{i\omega x} n_t(x)\mathrm{d}x$$

$$\hat{p}_t(\omega) = \int_{-\infty}^{+\infty} e^{i\omega x} p_t(x)\mathrm{d}x$$

where $i = \sqrt{-1}$. Then taking the Fourier transform of linearised equations (6.13) we get

$$\begin{bmatrix}\hat{n}_{t+1}(\omega)\\ \hat{p}_{t+1}(\omega)\end{bmatrix} = \mathbf{KJ}\begin{bmatrix}\hat{n}_t(\omega)\\ \hat{p}_t(\omega)\end{bmatrix}$$

where $\mathbf{K}$ is given by

$$\mathbf{K} = \begin{bmatrix}\hat{k}_N(\omega) & 0\\ 0 & \hat{k}_P(\omega)\end{bmatrix}.$$

Now for $j \in \{N, P\}$

$$\begin{aligned}
\hat{k}_j(\omega) &= \int_\Omega k_j(x) e^{i\omega x} \mathrm{d}x \\
&= (1-\mu_j) \int_\Omega \delta(x) I(|x|) e^{i\omega x} \mathrm{d}x + \frac{\mu_j}{2} \int_\Omega \delta(x+1/n) I(|x|) e^{i\omega x} \mathrm{d}x \\
&\quad + \frac{\mu_j}{2} \int_\Omega \delta(x-1/n) I(|x|) e^{i\omega x} \mathrm{d}x \\
&= 1 - \mu_j + \frac{\mu_j}{2} e^{-\frac{i\omega}{n}} \int_\Omega \delta(y) I(|y-1/n|) e^{i\omega y} \mathrm{d}y \\
&\quad + \frac{\mu_j}{2} e^{\frac{i\omega}{n}} \int_\Omega \delta(y) I(|y+1/n|) e^{i\omega y} \mathrm{d}y \\
&= 1 - \mu_j + \frac{\mu_j}{2} \left(e^{-\frac{i\omega}{n}} + e^{\frac{i\omega}{n}} \right) \\
&= 1 - \mu_j + \mu_j \cosh\left(i \frac{\omega}{n} \right) \\
&= 1 - \mu_j + \mu_j \cos\left(\frac{\omega}{n} \right).
\end{aligned}$$

Now, in the absence of dispersal $\mathbf{K} = \mathbf{I}$ and under the assumption that (N^*, P^*) is linearly stable, it follows that all the eigenvalues of $\mathbf{J}$ have modulus less than one. Hence, a dispersal-driven instability can arise if, and only if, the matrix $\mathbf{KJ}$ has one or more eigenvalues with modulus greater than one. Now by the Jury conditions the local dynamics are linearly stable if, and only if,

$$1 - \mathrm{tr}(\mathbf{J}) + \det(\mathbf{J}) > 0 \tag{6.14a}$$

$$1 + \mathrm{tr}(\mathbf{J}) + \det(\mathbf{J}) > 0 \tag{6.14b}$$

$$1 - \det(\mathbf{J}) > 0. \tag{6.14c}$$

Hence adding (6.14a) and (6.14b) and combining with (6.14c) yields

$$|\det(\mathbf{J})| < 1.$$

Note that $|\hat{k}_j| \leq 1$ for $j \in \{N, P\}$, and therefore

$$\begin{aligned}
|\det(\mathbf{KJ})| &= \left| \hat{k}_N \hat{k}_P \det(\mathbf{J}) \right| \\
&< 1.
\end{aligned}$$

Hence by the Jury conditions the result follows. □

Notice that the matrix $\mathbf{K}$ depends on the wave number ω, and hence dispersal-driven instability will occur (it at all) for limited ranges of ω.
Inequality (6.12a) guarantees that all real eigenvalues of $\mathbf{KJ}$ are less than $+1$. If this inequality is violated then the spatially homogeneous steady state will lose stability, and

the solution will become spatially structured. This bifurcation is known as a plus-one bifurcation. Inequality (6.12b) guarantees that all real eigenvalues of **KJ** are greater than -1. If this inequality is violated then the spatially homogeneous steady state will lose stability, and the solution will become spatially structured and time-periodic with period two. This type of bifurcation is known as a minus-one bifurcation.

Neubert et al. (1995) are able to show that for integro-difference equations that exploiter-victim type of dynamics are necessary for dispersal-driven instabilities. Therefore, it may be possible to get dispersal-driven instabilities for the host-pathogen model studied in Chapter 5. However, there can be no dispersal-driven instabilities for competition models which is consistent with the Hassell formulation (Rohani et al. 1996).

In this model formulation, I have assumed that the species only disperse to the neighbouring patches. However, with a simple adjustment to the functions k_N and k_P, more complex dispersal structures may be used. For example, one could easily modify the above analysis to incorporate movement to further patches, or to include unequal weighting to represent wind effects or olfactory cues.

By way of an example I will demonstrate this theory on a discrete-time predator-prey model

$$N_{t+1} = N_t e^{r(1-N_t-P_t)} \tag{6.15a}$$

$$P_{t+1} = cN_tP_t \tag{6.15b}$$

where N_t and P_t are the densities of the prey and predators at generation t. The dynamics of this system are not important for the purpose of this example, other than this model exhibits exploiter-victim type dynamics.

Model 6.15 has a unique positive steady state

$$N^* = \frac{1}{c}$$

$$P^* = 1 - \frac{1}{c}$$

which is biologically realistic if $c > 1$ and is linearly stable if, and only if,

$$1 < c < 2$$

$$0 < r < \frac{4c}{3-c}.$$

Now for a plus-one bifurcation inequality (6.12a) must be violated. Thus we may plot

$$Q = 1 - \mathrm{tr}(\mathbf{KJ}) + \det(\mathbf{KJ})$$

as a function of ω. If $Q(\omega) < 0$ for some value of ω then dispersion will have destabilised the homogeneous steady state, giving rise to a spatially structured solution.

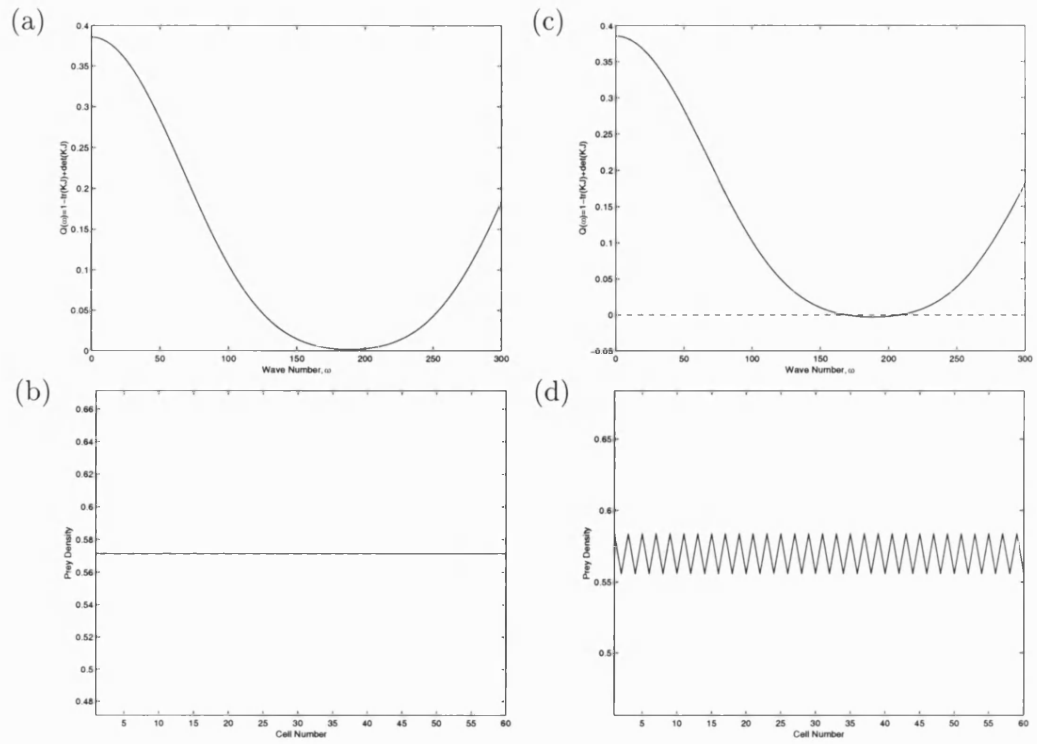


Figure 6-4: A plus-one bifurcation for a 1D CML. In (a) the function $Q(\omega) > 0$ thus the CML in (b) has a spatially uniform steady state. In (c) the function $Q(\omega) < 0$ for some interval of ω. Therefore there is a plus-one bifurcation, which is seen in the CML (d) where the solution is spatially structure. The simulations have been run for 10000 generations from random initial conditions. The parameter values for each plot are $n = 60$, $r = 0.9$, $c = 1.75$, $\mu_P = 0.09$ and in (a) & (b) $\mu_N = 0.89$ and in (c) & (d) $\mu_N = 0.90$.

In Figure 6-4 we see that by increasing the fraction of prey dispersing the uniform steady state is destabilised. This dispersal-driven instability occurs because the prey species disperses so quickly that the predators are unable to keep up and exert their stabilising influence on the prey which is consistent with reaction-diffusion equations (Segel & Jackson 1972).
I now turn my attention to two spatial dimensions.

The 2D CML

The formulation of the 2D CML is much the same as the 1D case. I assume that each species can disperse to it's eight nearest neighbouring lattice points with equal weighting and that there are $n \times n$ lattice points. Stability conditions are similar to that of the 1D case and the analysis depends on the 2D integro-difference model with

periodic boundary conditions:

$$N_{t+1}(x,y) = \int_\Omega\int_\Omega k_N(x-u,y-v)f(N_t(u,v),P_t(u,v))\mathrm{d}u\mathrm{d}v \tag{6.16a}$$

$$P_{t+1}(x,y) = \int_\Omega\int_\Omega k_P(x-u,y-v)g(N_t(u,v),P_t(u,v))\mathrm{d}u\mathrm{d}v \tag{6.16b}$$

where $(x,y)\in[0,1]\times[0,1]=\Omega\times\Omega$ and the redistribution kernels are given by

$$\begin{aligned}k_j(x-u,y-v) =& \Big[(1-\mu_j)\delta(x-u,y-v)+\frac{\mu_j}{8}\Big\{\delta(x-u+1/n,y-v)\\ &+\delta(x-u-1/n,y-v)+\delta(x-u,y-v+1/n)\\ &+\delta(x-u,y-v-1/n)+\delta(x-u+1/n,y-v+1/n)\\ &+\delta(x-u-1/n,y-v-1/n)+\delta(x-u+1/n,y-v-1/n)\\ &+\delta(x-u-1/n,y-v+1/n)\Big\}\Big]I(|x-u|,|y-v|)\end{aligned}$$

for $j\in\{N,P\}$, where $\delta(\cdot,\cdot)$ is the 2D Dirac delta function, and

$$I(|x-u,y-v|)=\begin{cases}1 & \text{if } (|x-u|,|y-v|)\in\mathcal{L}\times\mathcal{L}\\ 0 & \text{otherwise}\end{cases}$$

is the indicator function, and $\mathcal{L}=\{0,1/n,2/n,\ldots,1\}$ is the set of lattice points.

Lemma 6.2.3. *For the two interacting species model* (6.8)*, the spatially extended two dimensional CML or equivalently* (6.16)*, can be destabilised by dispersion if, and only if, one or more of the following inequalities are violated:*

$$1-\mathrm{tr}(\mathbf{KJ})+\det(\mathbf{KJ}) > 0 \tag{6.17a}$$

$$1+\mathrm{tr}(\mathbf{KJ})+\det(\mathbf{KJ}) > 0 \tag{6.17b}$$

where $\mathrm{tr}(\cdot)$ *and* $\det(\cdot)$ *are the trace and determinant of the matrix respectively,*

$$\mathbf{K}=\begin{bmatrix}\hat{k}_N & 0\\ 0 & \hat{k}_P\end{bmatrix}$$

where

$$\hat{k}_j(\omega_1,\omega_2)=1-\mu_j+\frac{\mu_j}{4}\left[\cos\left(\frac{\omega_1}{n}\right)+\cos\left(\frac{\omega_2}{n}\right)+2\cos\left(\frac{\omega_1}{n}\right)\cos\left(\frac{\omega_2}{n}\right)\right],$$

and $n\times n$ *is the number of lattice points in the CML,* ω_1,ω_2 *are the wave numbers, and* $\mathbf{J}$ *is the Jacobian of* (6.8)*.*

Proof. The proof is similar to that of Lemma 6.2.2. The only extra information required

is the two dimensional Fourier transform

$$\hat{f}(\omega_1, \omega_2) = \int_\Omega \int_\Omega f(x, y) e^{i(\omega_1 x + \omega_2 y)} \mathrm{d}x\mathrm{d}y.$$

The result then follows. □

I now demonstrate this numerically using the previous example (6.15).

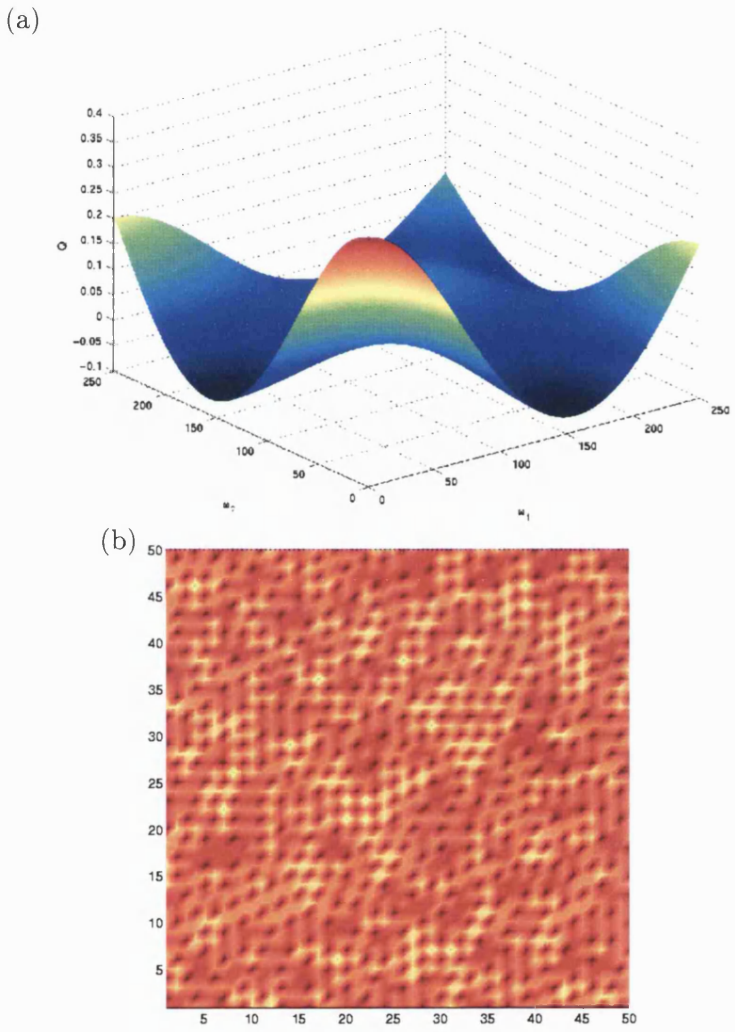


Figure 6-5: A plus-one bifurcation for a 2D CML. The model parameters are $r = 0.9$, $c = 1.75$, $n = 50$, $\mu_N = 0.97$ and $\mu_P = 0.05$. In (a) the figure shows a plot of $Q = 1 - \mathrm{tr}(\mathbf{KJ}) + \det(\mathbf{KJ})$ for varying values of ω_1 and ω_2. In (b) the figure shows the corresponding CML, which is simulated with random initial conditions.

In Figure 6-5 (a) I plot

$$Q = 1 - \mathrm{tr}(\mathbf{KJ}) + \det(\mathbf{KJ})$$

for varying values of ω_1 and ω_2. Clearly $Q < 0$ for some values of ω_1 and ω_2 and

thus there is a plus-one bifurcation, which leads to spatially structured solutions in the CML. This is shown in Figure 6-5 (b). To observe this type of dispersal-driven instability it is necessary that the prey have much a larger dispersion coefficient than the predators, so that the predators fail to have a stabilising effect on the prey. Failure to satisfy this condition leads to a spatially uniform solution.

6.2.3 Model 5.2 Spatially Extended - Dispersal-Driven Instabilities

I now consider the Host-Pathogen model considered in Chapter 5. Recall that the model is

$$N_{t+1} = A_1 p N_t e^{-r_1 N_t} f(\Phi_t) + A_2(1-p) N_t e^{-r_2 N_t} \tag{6.18a}$$

$$\Phi_{t+1} = \xi p N_t (1 - f(\Phi_t)) + \alpha \Phi_t + a \tag{6.18b}$$

where N_t and Φ_t are the densities of hosts and free-living pathogen at generation t, and

$$f(\Phi_t) = e^{-\beta \Phi_t}. \tag{6.18c}$$

Note that I have changed the parameter ω to ξ so that there is no confusion between the average number of spores released and the wave number.
In this section I consider whether Model 6.18 can give rise to dispersal-driven instabilities. To do this I only consider one spatial dimension so that the analysis is less complex, however conclusions derived for the 1D case can be extended to 2D.
As the above analysis shows, dispersal-driven instabilities can only occur if the local population model has exploiter-victim dynamics. That is,

$$J_{12} J_{21} < 0$$

where J_{ij} are elements of the Jacobian. For Model 6.18 we have

$$\begin{aligned}
J_{11} &= (1 - r_1 N^*) A_1 p e^{-r_1 N^*} e^{-\beta \Phi^*} + (1 - r_2 N^*) A_2 (1-p) e^{-r_2 N^*} \\
J_{12} &= -\beta A_1 p N^* e^{-r_1 N^*} e^{-\beta \Phi^*} < 0 \\
J_{21} &= \omega p \left(1 - e^{-\beta \Phi^*}\right) > 0 \\
J_{22} &= \beta \omega p N^* e^{-\beta \Phi^*} + \alpha.
\end{aligned}$$

Thus clearly $J_{12} J_{21} < 0$ and so it may be possible to get dispersal-driven instabilities. Recall that to get these instabilities we require to violate either (6.12a) or (6.12b).
I now consider general conditions for a plus-one bifurcation.
A plus-one bifurcation in a 1D CML happens when the local population dynamics are

stable and (6.12a) is violated, i.e.

$$\begin{aligned} Q(\omega) &= 1 - \mathrm{tr}(\mathbf{KJ}) + \det(\mathbf{KJ}) \\ &= 1 - (\hat{k}_N J_{11} + \hat{k}_\Phi J_{22}) + \hat{k}_N \hat{k}_\Phi \det(\mathbf{J}) < 0 \end{aligned}$$

where

$$\hat{k}_j = 1 - \mu_j + \mu_j \cos\left(\frac{\omega}{n}\right).$$

Now, we may rewrite Q as a quadratic in cos to get

$$Q(\omega) = A\cos^2\left(\frac{\omega}{n}\right) + B\cos\left(\frac{\omega}{n}\right) + C \tag{6.19}$$

where

$$\begin{aligned} A &= \mu_N \mu_\Phi \det(\mathbf{J}) \\ B &= -\mu_N J_{11} - \mu_\Phi J_{22} + \{(1-\mu_N)\mu_\Phi + (1-\mu_\Phi)\mu_N\}\det(\mathbf{J}) \\ C &= 1 - (1-\mu_N)J_{11} - (1-\mu_\Phi)J_{22} + (1-\mu_N)(1-\mu_\Phi)\det(\mathbf{J}). \end{aligned}$$

Now

$$Q(0) = 1 - \mathrm{tr}(\mathbf{J}) + \det(\mathbf{J}) > 0$$

since we assume that the local dynamics are stable. Also since Q is a quadratic in cos, Q must be periodic. Hence, if a plus-one bifurcation is possible by varying the dispersal coefficients, the point at which $Q(\omega)$ first crosses the ω-axis, ω^*, is given by simultaneously solving

$$Q(\omega^*) = 0 \quad \text{and} \quad \frac{dQ}{d\omega}(\omega^*) = 0. \tag{6.20}$$

Now

$$\frac{dQ}{d\omega} = -\frac{2A}{n}\cos\left(\frac{\omega}{n}\right)\sin\left(\frac{\omega}{n}\right) - \frac{B}{n}\sin\left(\frac{\omega}{n}\right),$$

and so $\frac{dQ}{d\omega}(\omega) = 0$ if, and only if,

$$\sin\left(\frac{\omega}{n}\right) = 0 \quad \text{or} \quad \cos\left(\frac{\omega}{n}\right) = -\frac{B}{2A}. \tag{6.21}$$

The period of Q is $2n\pi$, since $Q(0) = Q(2n\pi) > 0$. Hence there are only 3 possible solutions to (6.21):

1. If $0 < -\dfrac{B}{2A} < 1$ then $\omega = n\arccos\left(-\dfrac{B}{2A}\right) < \dfrac{n\pi}{2}$. Hence there exists an ω^* satisfying (6.20) if $B^2 - 4AC = 0$ where $0 < \omega^* < \dfrac{n\pi}{2}$.

2. If $-1 < -\dfrac{B}{2A} < 0$ then $\dfrac{n\pi}{2} < \omega = n\arccos\left(-\dfrac{B}{2A}\right) < n\pi$. Hence there exists an

ω^* satisfying (6.20) if $B^2 - 4AC = 0$ where $n\pi/2 < \omega^* < n\pi$.

3. If $\left|\frac{B}{2A}\right| > 1$ then $\omega = n\pi$. Hence there exists an ω^* satisfying (6.20) if $A-B+C = 0$.

The value ω^* predicts the wavelength of the spatially structured solution close to the bifurcation. Since I have assumed in the CML formulation that the length of the domain is one, it follows that there will be $\omega^*/2\pi$ troughs in the spatially structured solution. Clearly if $\omega^* = n\pi$ (as in case 3 above) then there will be $n/2$ troughs in the spatially structured solution, which means that the values will alternate between two values in the 1D CML (as in Figure 6-4). This is the maximum number of troughs possible.

For a minus-one bifurcation a similar result holds. This time the function we are interested in is

$$Q(\omega) = 1 + \mathrm{tr}(\mathbf{KJ}) + \det(\mathbf{KJ})$$

which can be written as

$$Q(\omega) = A\cos^2\left(\frac{\omega}{n}\right) + B\cos\left(\frac{\omega}{n}\right) + C \tag{6.22}$$

where

$$\begin{aligned} A &= \mu_N\mu_\Phi\det(\mathbf{J}) \\ B &= \mu_N J_{11} + \mu_\Phi J_{22} + \{(1-\mu_N)\mu_\Phi + (1-\mu_\Phi)\mu_N\}\det(\mathbf{J}) \\ C &= 1 + (1-\mu_N)J_{11} + (1-\mu_\Phi)J_{22} + (1-\mu_N)(1-\mu_\Phi)\det(\mathbf{J}). \end{aligned}$$

Then using the above analysis the result follows except the solution will also be time-dependent and have period 2.

I now apply this result to Model 6.18 and discuss some of its implications.

In Figure 6-6 I fix the model parameters for Model 6.18 and vary the dispersion coefficients, μ_N and μ_Φ. One can see that in graphs (a), (b) and (c), where host dispersal is large and pathogen dispersal is small then the function (6.22) becomes negative for some wave number interval, whilst (6.19) remains positive. Hence one would expect to observe a minus-one bifurcation, which gives period two time-dependent spatially structured solution in the CML. This is shown in (c) where successive snapshots of the CML simulation have been shown after a transient period of 1000 generations (the solid and dashed lines). In (d), (e) and (f), where there is little difference in the two dispersion coefficients, we see that both the functions (6.22) and (6.19) are positive for all wave numbers. Hence the CML has a spatially uniform solution. In (g), (h) and (i), where host dispersal is small and pathogen dispersal is large then the function

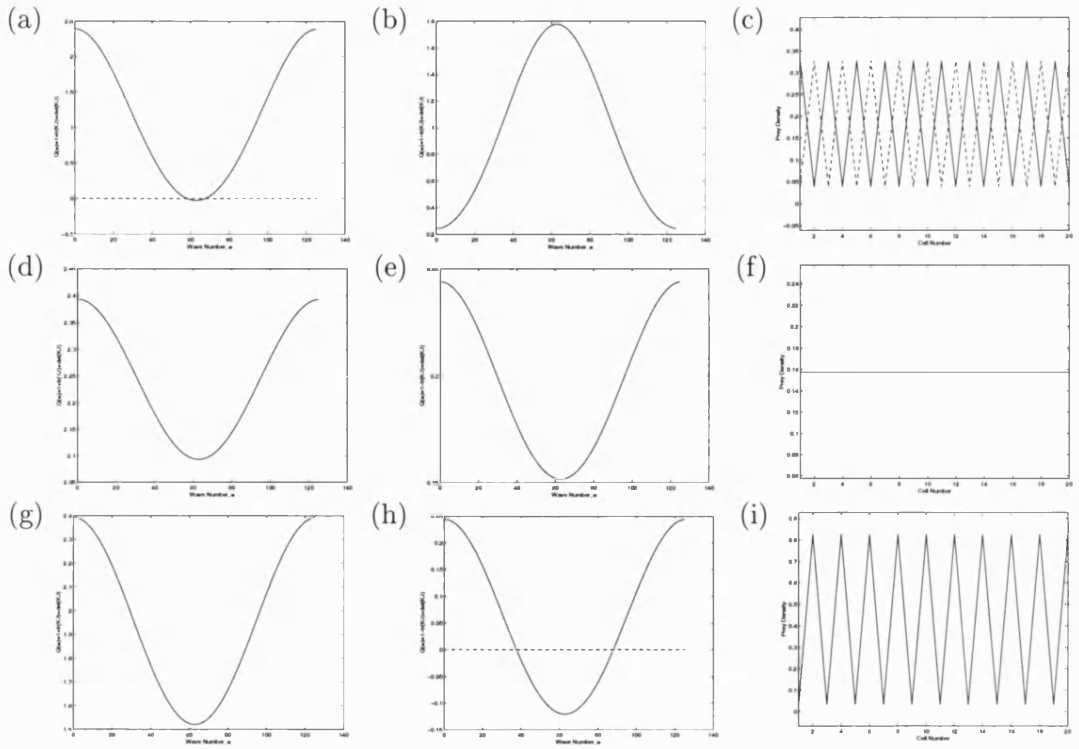


Figure 6-6: Dispersal-driven instabilities. The local host-pathogen parameter values for Model 6.18 are $A_1 = 5.6$, $A_2 = 2.59$, $r_1 = 0.03$, $r_2 = 2$, $\beta = 1.78$, $\xi = 1.07$, $\alpha = 0.06$, $p = 0.95$ and $a = 0.8$. For these parameter values Model 6.18 has a unique positive linearly stable equilibrium value. In (a), (b) & (c) the dispersion coefficients are $\mu_N = 0.99$ and $\mu_\Phi = 0.3$. In (d), (e) & (f) the dispersion coefficients are $\mu_N = 0.02$ and $\mu_\Phi = 0.3$. In (g), (h) & (i) the dispersion coefficients are $\mu_N = 0.02$ and $\mu_\Phi = 0.99$. Graphs (a), (d) & (g) are plots of the function (6.22), hence if the function becomes negative for some wave number then a minus-one bifurcation will be observed. Graphs (b), (e) & (h) are plots of the function (6.19), hence if the function becomes negative for some wave number then a plus-one bifurcation will be observed. Graphs (c), (f) & (i) are the resultant CML simulations. The CML simulations are started from random initial population values and are run for a transient period of 1000 generations. See text for more detail.

(6.19) becomes negative for some wave number interval, whilst (6.22) remains positive. Hence the CML exhibits a spatially structured solution that is stationary in time, since a plus-one bifurcation has occurred.

In Figure 6-7 we see that as the amount of externally applied pathogen is increased the regions in (μ_N, μ_Φ)-space where dispersal-driven instabilities occur become smaller. If a is increased sufficiently then the regions vanish as the host population dies-out and therefore no dispersal-driven instabilities can occur.

In Figure 6-8 we see that the number of lattice points makes no difference to the regions in (μ_N, μ_Φ)-space where dispersal-driven instabilities occur (compare to Figure 6-

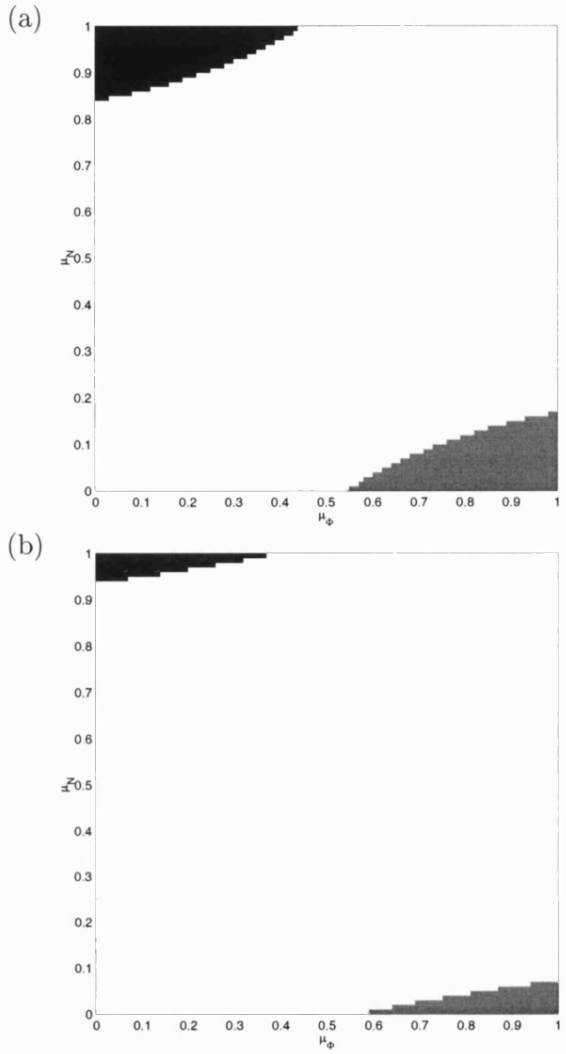


Figure 6-7: Dispersal-driven instabilities in Model 6.18. The areas shaded in black correspond to regions in (μ_N, μ_Φ)-space where a minus-one bifurcation occurs, so that the spatial solution is spatially structured and time-dependent. The areas in gray correspond to regions in (μ_N, μ_Φ)-space where a plus-one bifurcation occurs, so that the spatial solution is spatially structured and time-independent. In both regions $|B/2A| > 1$, and so the number of troughs is half the number of lattice points. The local host-pathogen parameter values for Model 6.18 are $A_1 = 5.6$, $A_2 = 2.59$, $r_1 = 0.03$, $r_2 = 2$, $\beta = 1.78$, $\xi = 1.07$, $\alpha = 0.06$, $p = 0.95$ and in (a) $a = 0.5$ and in (b) $a = 0.8$. The 1D CML has 60 lattice points.

7 (b)). This obvious from observing the functions (6.19) and (6.22) which determine if dispersal-driven instabilities are possible. This is because the number of lattice points, n, determines the frequency of the functions (6.19) and (6.22), which gives the number of troughs in the CML, whilst the coefficients A, B, and C (which depend on the model parameters) determine the amplitude of the functions.

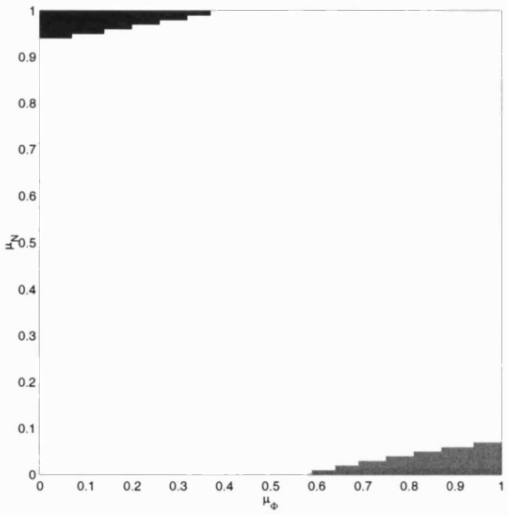


Figure 6-8: Dispersal-driven instabilities in Model 6.18. The areas shaded in black correspond to regions in (μ_N, μ_Φ)-space where a minus-one bifurcation occurs, so that the spatial solution is spatially structured and time-dependent. The areas in gray correspond to regions in (μ_N, μ_Φ)-space where a plus-one bifurcation occurs, so that the spatial solution is spatially structured and time-independent. In both regions $|B/2A| > 1$, and so the number of troughs is half the number of lattice points. The local host-pathogen parameter values for Model 6.18 are the same as in Figure 6-7 (b). The 1D CML has 20 lattice points.

6.3 Spatially Structured Solutions With Unstable Local Dynamics

So far I have only discussed dispersal-driven instabilities. What if the local temporal dynamics are unstable? What effects do the dispersal coefficients have on the spatial dynamics?

Suppose that the local dynamics are unstable, so that in the absence of dispersal the host and pathogen densities oscillate in time. I now investigate the behaviour of the CML when these unstable dynamics are spatially extended.

From the extensive simulations that I have carried out on Model 6.18, I have classified the behaviours into two categories:

Spiral Waves: The hosts and pathogens form waves that sweep across the lattice that roughly repeat themselves in an ordered way. When plotting the total density of hosts against the total density of pathogen in a phase-plot, the results look very similar to that of limit cycles (see Figure 6-9). Due to the periodic boundary conditions the patterns that are observed are symmetric. When using smaller domains, the spirals may not be observed so clearly, and may result in 'periodic waves' that have similar properties as the spiral waves. The basis for the spiral

wave is founded on the propagation of a wave, in which the hosts invade the (almost) empty space which is then attacked by the pathogen, and then die out. This is clearly shown in Figure 6-10 where we see that the pathogen wave leads the host wave, so that the density of hosts is low in front of the host wave.

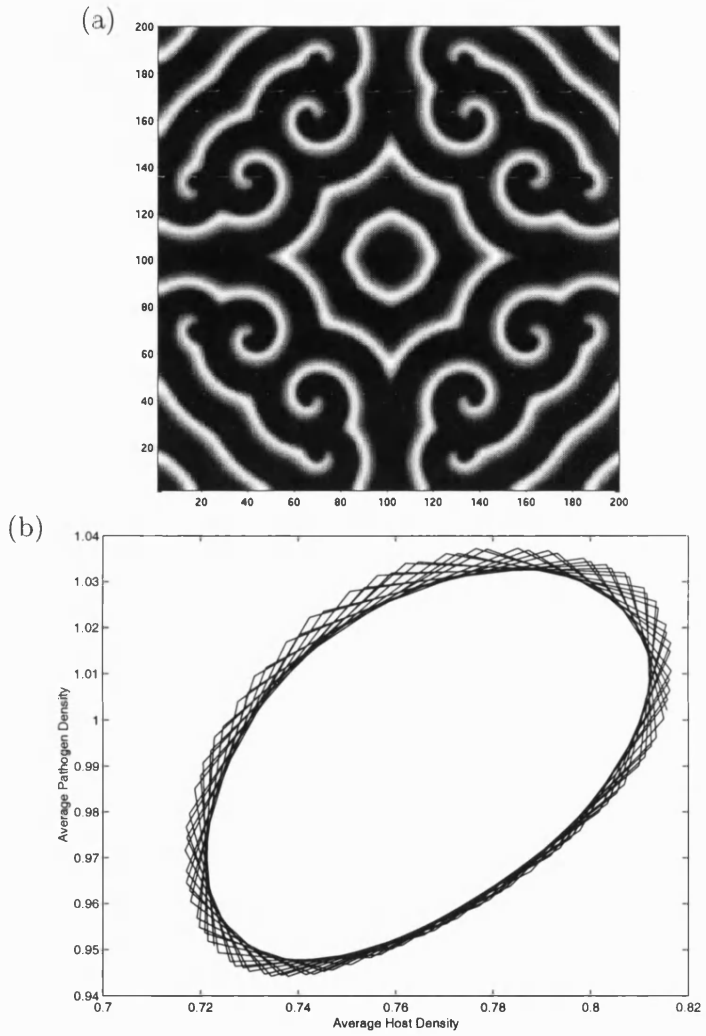


Figure 6-9: Spiral waves. The local host-pathogen parameter values for Model 6.18 are $A_1 = 5.6$, $A_2 = 2.59$, $r_1 = 0.03$, $r_2 = 2$, $\beta = 1.78$, $\xi = 1.07$, $a = 0.3$, $\alpha = 0.06$ and $p = 0.95$. The dispersion coefficients are $\mu_N = 0.5$ and $\mu_\Phi = 0.9$. There are 200×200 lattice points. The white areas denote where there are high densities of hosts, and black areas denote low densities. Initially the simulation had a positive host population at position $(1, 1)$ on the lattice and zero elsewhere. The CML snapshot is taken after a transient period of 1000 generations. The phase plot in figure (b) is the average host density per lattice point plotted against the average pathogen density. The phase plot is taken after a transient period of 900 generations, and run for a further 100 generations.

Stable Patterns: After a transient period the CML settles down to a spatially struc-

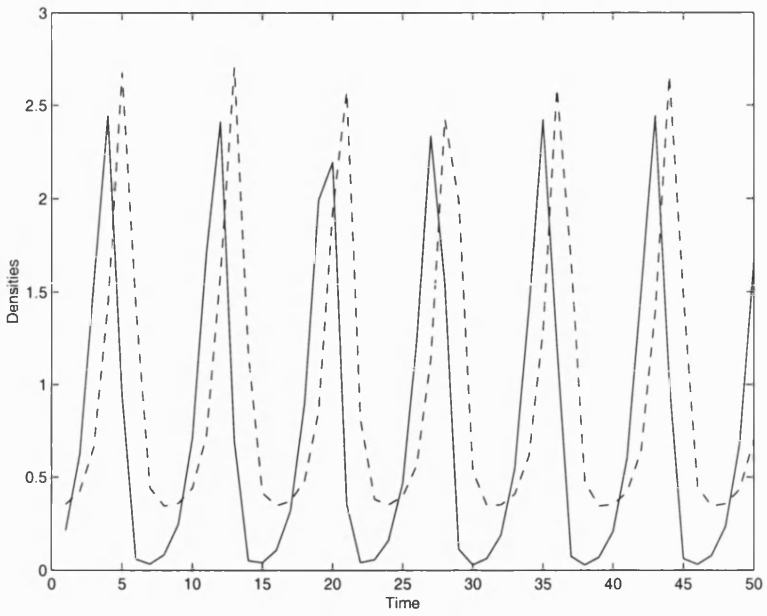


Figure 6-10: Wave Propagation. The local host-pathogen parameter values for Model 6.18 are $A_1 = 5.6$, $A_2 = 2.59$, $r_1 = 0.03$, $r_2 = 2$, $\beta = 1.78$, $\xi = 1.07$, $a = 0.3$, $\alpha = 0.06$ and $p = 0.95$. The dispersion coefficients are $\mu_N = 0.5$ and $\mu_\Phi = 0.9$. There are 50×50 lattice points. Initially the simulation had a positive host population at position $(1, 1)$ on the lattice and zero elsewhere. There is a transient period of 1000 generations before records are kept. The solid line and dashed line denote the densities of hosts and pathogen at position $(1, 1)$ on the lattice respectively.

tured pattern that remains stationary over time, as opposed to the spiral waves case. The patterns usually have patches where there is a high density of hosts (see Figure 6-11). In general, where the hosts have high densities, the pathogen is at low density and vice versa. One can clearly see in the phase plot the densities of hosts and pathogen tend to a stable value over time, as indicated in the top right corner of the plot, where the host-pathogen densities spiral in to a fixed point. Thus the average host-pathogen densities tend to a constant value over time, as opposed to the oscillatory behaviour seen in the spiral waves case.

In Comins et al. (1992) the authors discuss another type of behaviour that they call spatial chaos. In this case the host and parasitoid (in my case a pathogen) populations fluctuate from patch to patch with no long-term spatial organisation. However, I do not observe these phenomenon with my CML and model. There are a few notable differences between the model presented in Comins et al. (1992) and the model presented here:

- The authors in the Comins et al. (1992) paper consider the Nicholson-Bailey model which is inherently more unstable than my model. However, I do not see

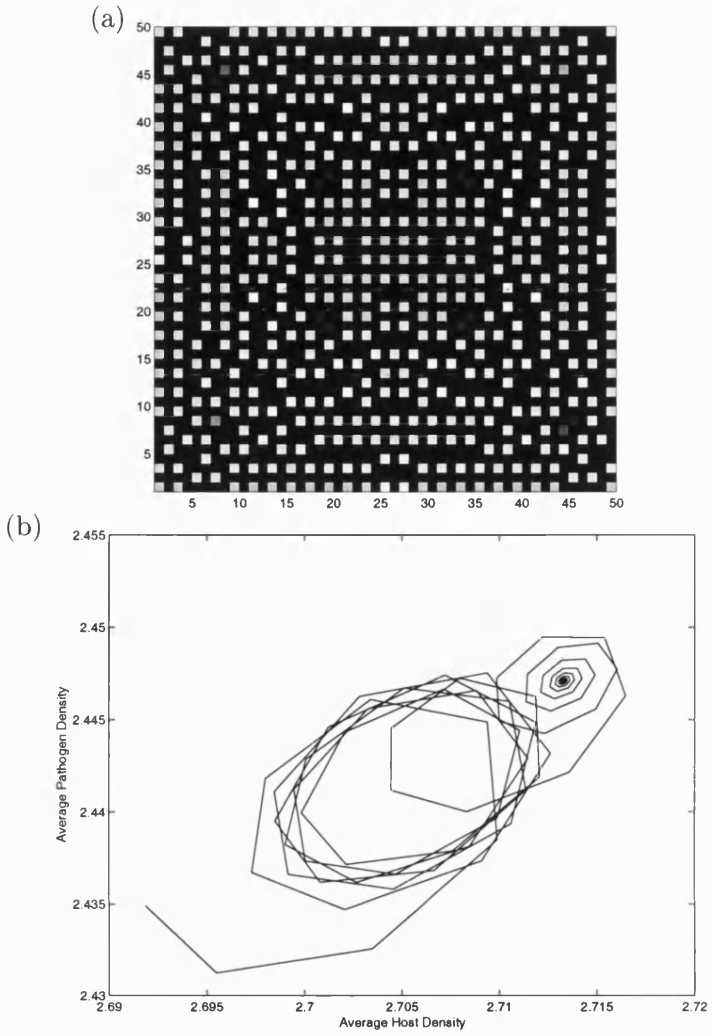


Figure 6-11: Stable Patterns. The local host-pathogen parameter values for Model 6.18 are the same as in Figure 6-9. The dispersion coefficients are $\mu_N = 0.01$ and $\mu_\Phi = 0.98$. The CML has 50×50 lattice points, and the initial conditions are as in Figure 6-9. The CML in figure (a) are of the host population densities. Lattice points that are coded in white denote areas of high population density and areas coded in black denotes areas of low density. The CML snapshot is taken after a transient period of 1000 generations. The phase plot in figure (b) is the average host density per lattice point plotted against the average pathogen density. The phase plot is taken after a transient period of 400 generations, and run for a further 200 generations. Initially the simulation had a positive host population at position $(1, 1)$ on the lattice and zero elsewhere.

this to cause any differences in the types of behaviours observed as my model can be driven unstable by suitable choices of parameters.

- The authors in the Comins et al. (1992) paper use reflective boundary conditions, where I use periodic boundary conditions. I claim that this difference is the reason

as to why I do not observe the spatial chaos. This is due to the symmetry that the periodic boundary conditions impose. I have also carried out extensive numerical simulations on my model with absorbing boundary conditions and found that I could observe spatial chaos, as shown in Figure 6-12, where identical simulations are run on the CML but with differing boundary conditions. It is clear that in both the CML snapshots, (a) and (c), that the spatial patterning is complex spiral waves, however, in the figure (c) the patterning is symmetric due to the periodic boundary conditions. When examining the phase diagrams in figures (b) and (d), we clearly see that the CML with absorbing boundary conditions is more chaotic than the CML with the periodic boundary conditions.

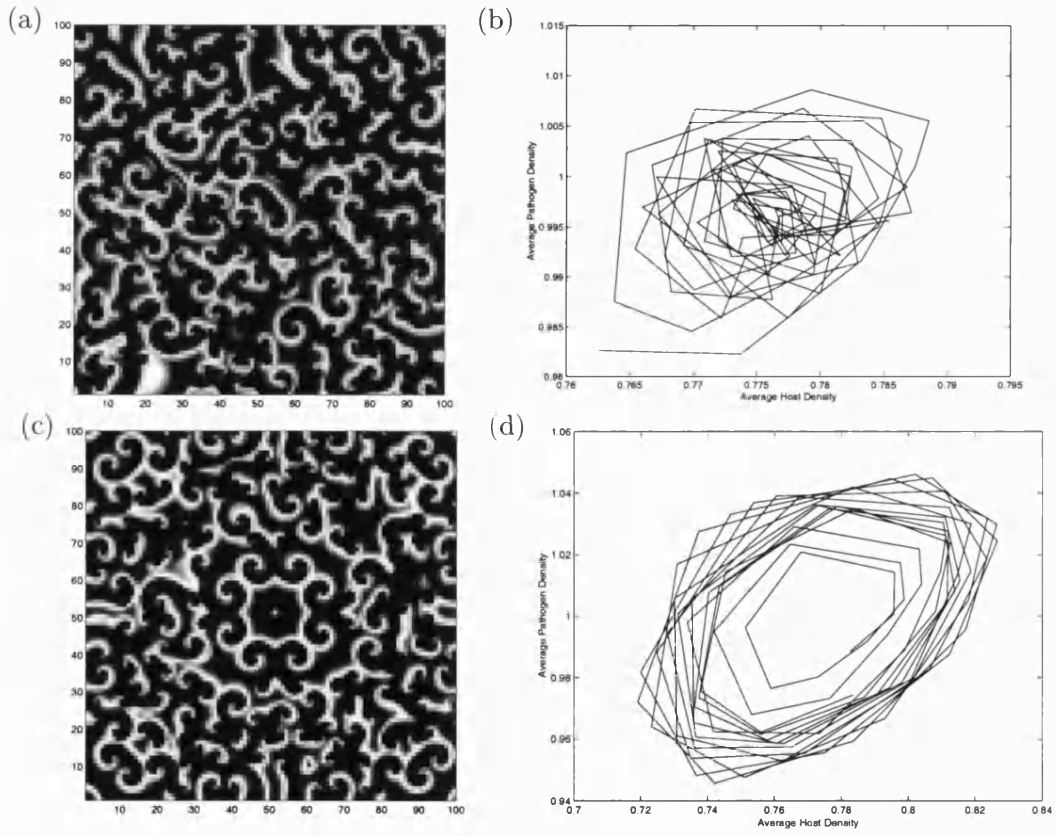


Figure 6-12: Spatial chaos. The local host-pathogen parameter values for Model 6.18 are the same as in Figure 6-9, with identical initial conditions. The dispersion coefficients are $\mu_N = 0.1$ and $\mu_\Phi = 0.11$. In figures (a) and (b), the simulations have absorbing boundary conditions, as opposed to periodic boundary conditions in figures (c) and (d).

As the authors in the Comins et al. (1992) paper point out strictly speaking, the spiral waves case is still chaotic since the position and the number of focal points varies slowly with time in non-repeating patterns. This can be clearly seen in the phase plot

in Figure 6-9, where the phase is almost periodic, but not quite.
The size of the lattice does not seem to have a massive difference on the spatial structures observed. However, the spatial patterning in the spiral waves case does differ. In smaller lattice sizes it is often harder to see the spirals, but instead one observes 'periodic' regions of high and low densities of host and pathogen that vary over time. However, it is clear that these are still spiral waves since the phase plots are similar (see Figure 6-13).

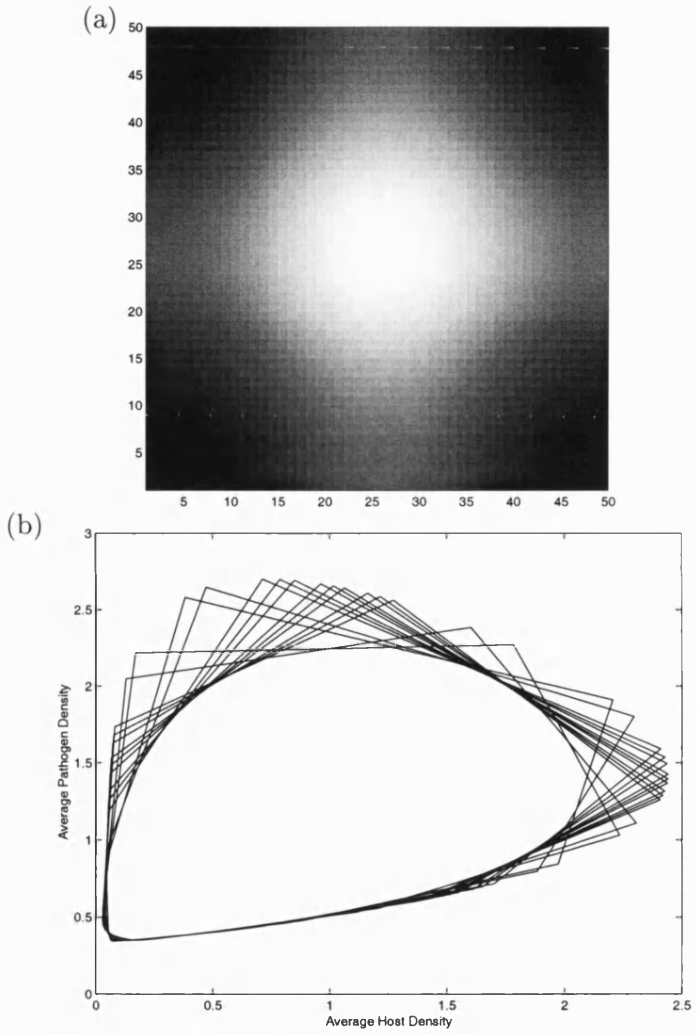


Figure 6-13: Spiral waves in a smaller lattice. The conditions and parameter values are identical to that in Figure 6-9 except that the lattice has 50 lattice points.

I have also observed that:

- The effects of increasing the average contributions to the next generation (A_i's) has the effect of making the spatial patterning more complex. This is due to the fact that as the average contribution to the next generation is increased, the local

population dynamics become more unstable in time. Thus producing more spiral waves that collide and produce more complex dynamics.

- An increase in the amount of externally applied pathogen (a) has the opposite effect. As shown in Chapter 5, an increase in the amount of externally applied pathogen has a stabilising effect on the host dynamics. Therefore, by increasing the amount of externally applied pathogen, one can stabilise the locally unstable dynamics and therefore prevent this type of spatial patterning. However, depending on the model parameters, the system may still show spatial patterning via dispersal-driven instabilities.

6.4 Conclusions & Discussion

In this chapter I have shown that the CML can be represented in the form of an integro-difference equation. For a general single species model, I have shown that the size of the CML has no effect on the spatial stability of the model which also does not depend on the dispersal coefficients. Therefore, this formulation of the CML seems like a biologically sensible idea.

To get dispersal-driven instabilities we need at least two interacting species, which is consistent with continuous-time reaction-diffusion models. I then derive general conditions for the instabilities, and demonstrate that they occur when there are large differences in the dispersal coefficients between the two species. Moreover, the instabilities will only occur when the species have an exploiter-victim type of relationship. The dispersal-driven instabilities are independent of the size of the CML.

From the numerous numerical simulations that I have carried out on Model 6.18, it seems that when the host dispersal coefficient is small and the pathogen coefficient is large, then if a dispersal-driven instability is possible, a plus-one bifurcation is always observed. If the dispersal coefficients are reversed then a minus-one bifurcation is always observed. It seems clear that dispersal-driven instabilities can only occur when the difference in the dispersal coefficients is large. This is required because the pathogen needs to lose its stabilising effect on the host.

Since the pathogen has a stabilising effect on the host population densities (as shown in Chapter 5), it is unsurprising that by increasing the amount of externally applied pathogen, a, the less probability there is of obtaining dispersal-driven instabilities.

For the numerical simulations that I have carried out, I have only been able to get bifurcations where the number of troughs is half the number of lattice points (the maximum number of troughs possible). I have been able to show (numerically) that the regions in (μ_N, μ_Φ)-space where a bifurcation is possible then $\left|\frac{B}{2A}\right| > 1$.

The results published in Winder et al. (2001) show that there is spatial patterning between a prey aphid species and a predatory beetle species. From the data collected,

the authors are able to show that the spatial patterning is not stationary but moves about the field, with the aphid species following the beetles. The analysis and simulations carried out in this chapter would suggest that the mechanism for this patterning comes from unstable temporal dynamics, rather than from dispersal-driven instabilities. One might expect to see this since aphids have massive reproductive potentials, and hence the average contribution to the next generation, A_i, will be large. As seen in Chapter 5, if the average contribution to the next generation is large then the local population dynamics are likely to be unstable, resulting in oscillating population densities.

The main results of this chapter rely on the fact that the CML may be written as an integro-difference equation with a special redistribution kernel. Due to this simple idea, many of the results obtained for integro-difference equations can now be applied to the CML. For example, many authors are interested in invasions of species or diseases, which are modelled by integro-difference equations (see Kot (1992), Hart & Gardner (1997) and Wang, Kot & Neubert (2002) for examples).

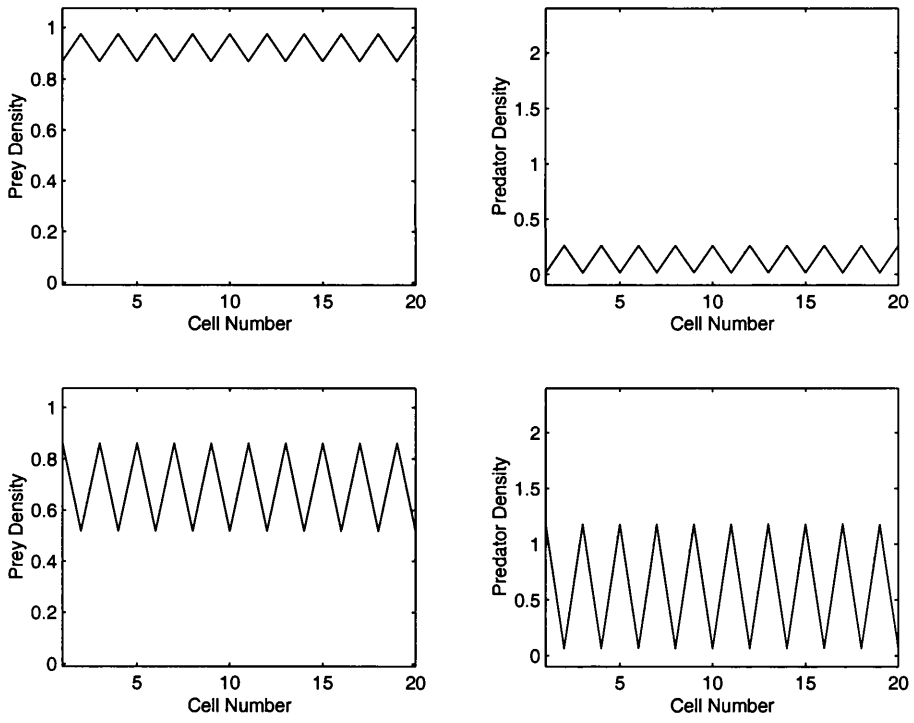


Figure 6-14: Second bifurcation. CML simulations of Model 6.15 with the model parameters $r = 0.9$, $c = 1.75$, $\mu_N = 0.02$ and $\mu_\Phi = 0.99$. The simulation is started from random initial conditions. The top set of figures are snapshots of the CML after 10000 generations. The bottom set of plots are snapshots of the same CML after 10004 generations.

For the example predator-prey model (6.15), numerics for the 1D CML have indicated

that a second bifurcation is possible where there are two periodic frequencies. Firstly from the minus-one bifurcation where the densities oscillate between neighbouring lattice points in successive generations (as in Figure 6-6 (c)), and a second frequency where the densities vary over time periodically (see Figure 6-14).
Figure 6-14 shows that more complex spatial patterning can be observed by dispersal-driven instabilities. However, this second bifurcation is currently not well understood and needs more analytical and numerical investigation, which I propose to do in the future.
In this chapter I have also considered what happens when locally unstable dynamics are spatially extended using the CML. The patterning observed depends on the size of the lattice, the boundary conditions and the dispersion coefficients. For periodic boundary conditions the types of behaviour observed can be generally classified into two categories; spiral waves and stable patterns, which can both be observed from identical local dynamics but differing dispersion coefficients. In the spiral waves case we see that (see Figure 6-9) the average host density fluctuates over time, and mimics the non-spatial model solution (see Figure 6-15).

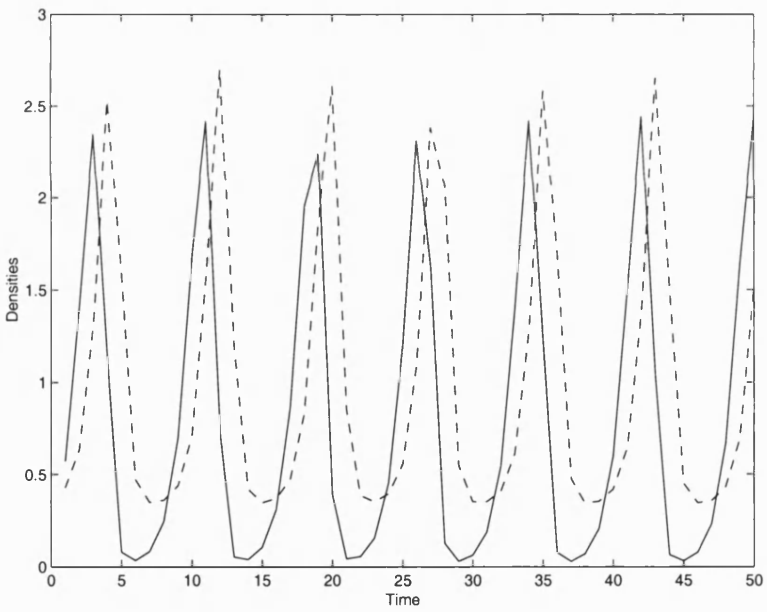


Figure 6-15: Non-spatial model solution of Model 6.18 with the model parameters identical to that in Figure 6-9 and Figure 6-11. The solid lines denote the host density and the dashed line denotes the pathogen density.

However, we can clearly see that this local behaviour is not mimicked in the stable patterns case, where the average host density goes to a fixed point (see Figure 6-11 (b)). Interestingly the fixed point ($N \approx 2.71$) is much larger than the time-averaged average host density in the spiral waves case ($N \approx 0.77$). Therefore, from a control

point of view, the stable patterns case is not desirable.

Chapter 7

Summary & Discussion

In this thesis I have investigated the role of resistance to controlled host-pathogen systems.

In Chapter 2 I considered a continuous-time model for two strains of aphid; one that is susceptible to the pathogen, and the other resistant to the pathogen. The interspecific competition between the the two strains is investigated in two cases; when no pathogen is externally applied, and when the pathogen is externally applied. The presence of the resistant strain means that total eradication of the aphid populations is not possible, no matter how much of the pathogen is applied to the system. However, the interspecific competition between the two strains plays an important part in the possible eradication of one of the species. By adjusting the amount of externally applied pathogen, it may be possible to control the pest insect levels in the field, since the susceptible aphids will always decrease in density, which may allow the resistant aphids to increase in density. Therefore, damage to the crops by the feeding insects could be minimised by selecting appropriate levels of externally applied pathogen.

So far, many authors have only considered the trade-off between resistance and reproductive output (see Antonovics & Thrall (1994) and Bowers et al. (1994) for examples), however, these models do not take into account the differing competitive abilities. From the analysis carried out in Chapters 2 and 3, it is clear that there exists a trade-off between the ability to compete and the ability to defend from the attack of the pathogen. If both strains are strong interspecific competitors then the susceptible and resistant strains can persist if insufficient amounts of pathogen are applied. However, applying sufficient amounts of pathogen causes the susceptible strain to dwindle to eradication, whilst leaving the resistant strain to go to their environmental carrying capacity.

Perhaps the more interesting scenario is when the susceptible strain is a strong interspecific competitor and the resistant strain is a weak interspecific competitor. Then depending on the amount of externally applied pathogen, and the time between sprays, the resistant strain can out-compete the susceptible strain.

In Chapter 4 I assume that there exists a trade-off between resistance and reproductive

rate to show that depending on how costly the acquired resistance is determines the evolutionary behaviour. Moreover, by increasing the amount of externally applied pathogen, I have been able to show that it is more likely that the insects will evolve to a more resistant state. Exactly how this happens depends on the shape of the trade-off function. If the trade-off function is increasingly costly then a unique attracting singular strategy exists, which decreases as the amount of externally applied pathogen increases, meaning that the aphid population will evolve towards a more resistant state. However, if the trade-off function is decreasingly costly then depending on certain parameter constraints, the singular strategies are either repellors or branching points. By increasing the amount of externally applied pathogen the branching point vanishes since as the conditions for the insects become more harsh, there is less to be gained by the population becoming dimorphic, where one strain becomes more resistant and less fecund and the other strain becoming less resistant and more fecund. After the critical point the population can only remain monomorphic which evolve to a highly resistant state.

As shown in Chapters 2 & 3, there clearly exists a trade-off between resistance and intraspecific competitive ability. Using the technique of adaptive dynamics the evolution of this interspecific trait could be further explored.

Chapter 5 investigates a host-pathogen model in discrete time to represent the aphids ability to overwinter. Whilst the model results are similar to those presented in the continuous time model, I was able to show that the host-pathogen dynamics can be stabilised by an increase in the amount of externally applied pathogen. This is further shown in the model's spatial extension in Chapter 6. This is due to the inhibiting effect on the susceptible population.

Chapter 6 shows that incorporating space into the discrete-time model presented in Chapter 5 can have surprising effects. If the local dynamics are locally stable, then depending on the dispersion coefficients of the hosts and pathogen, dispersal-driven spatially structured distributions of the hosts may be possible. This could be important in pest management schemes where inspection of the crop for pests is vital, since a pest density sample may lead to false conclusions about the state of the pest problem, since the distribution of the pests may not be uniform.

If on the other hand, the local dynamics are unstable then applying the pathogen may have to be done carefully. Since if the pest insects are highly mobile (i.e. μ_N is large), then applying a pathogen that cannot easily disperse (i.e. μ_Φ is small) will lead to pest insect levels that are higher than the levels for a pathogen that can easily disperse. This result could be important when dealing with pathogens that rely on water droplets to disperse, since watering the crops will help with pathogen dispersal and hence rule out the possibility of stable spatial patterns forming which allow high pest insect densities. I have shown in Chapter 5 that by increasing the amount of externally applied pathogen

can have a stabilising effect on the local population dynamics. Therefore, it follows that increasing the amount of externally applied pathogen can have a stabilising effect on the spatial distribution of the insects. However, if the difference in the dispersion coefficients of the hosts and pathogen is large then spatial patterning may occur via a diffusion-driven instability.

7.1 Future Work

In all of the models presented in this thesis, I have assumed that the state of the crop remains constant. However, this may not be realistic for some species of aphid which cause serious amounts of damage to the plants. To model this, a new state variable would have to be introduced into the model equations that represent the state of the plants. The plant dynamics must depend on the density of hosts, and have a negative effect on the state of the plant as the density of insects increase. Moreover, as the state of the plant decreases, the dynamics of the insects must change. For example, since the plant is decreasing in quality, there maybe be effects on the hosts reproductive ability (i.e. a decrease in the intrinsic growth rate or a higher susceptibility to crowding) or pehaps an increase in the aphids dispersal, since low plant quality will mean that the aphids will not want to stay feeding on the plant.

In Chapter 4 I assume that there is a trade-off between susceptibility to the pathogen and intrinsic growth rate. However, as indicated in Chapters 2 and 3, there exists a trade-off in interspecific competitive ability. Firstly it would be interesting to explore the evolution resistance which has a trade-off with this competition trait. Then secondly to explore the evolution of resistance which has a simultaneous trade-off with both the intrinsic growth rate and the interspecific competitive ability.

In Chapter 5 I assumed that the probability of remaining susceptible at each generation, p, is constant. In future work it may be of interest to make this a function of the density of free-living pathogen. An interesting biological experiment would be to investigate the shape of this function and then try to fit a curve to the data. One might expect that as the amount of pathogen is increased, the probability of remaining susceptible at each generation will decrease. One possible suggestion is to use the funtion

$$p(\Phi_t) = e^{-\theta\Phi_t}$$

where θ is a coefficient of resistance. This function decreases as Φ_t increases, and has a range that lies in $[0, 1]$.

In Chapter 6 I investigate the stability of coupled map lattices for general systems of coupled two-species discrete-time models. To achieve this I transform the CML into an integro-difference equation via a special redistribution kernel. Therefore any analysis on integro-difference equations can now be applied to CML's, provided the indicator

function can be dealt with in the analysis.

As mentioned in Chapter 6, it is possible for some models to exhibit a second dispersal-driven instability, which allows the host population to be spatially patterned and to fluctuate periodically in time. So far I have been unable to simulate this result for the model presented in Chapter 5. Clearly further study is required in order to analyse this second bifurcation as it may be biologically important in terms of pest management.

Advances in molecular techniques have revealed that infections are often caused by multiple pathogen genotypes (see Hodson, Hitchman, Vanbergen, Hails, Hartley, Possee, Watt & Cory (2003) for example). Epidemiological mathematical models have tended to either avoid explicit consideration of mixed infections, or to assume within-host dynamics as a simple race between genotypes to gain the greatest share of host resources, as in the tragedy of the commons' hypothesis (Hardin 1968). In particular, models of mixed infections have not included obligate killers with external persistent infectious stages, as is the case with baculoviruses (Begon & Bowers 1995), this important omission being due to a lack of knowledge as to how mixed infections behave. We (myself, Prof. Philip Maini (Centre for Mathematical Biology, University of Oxford) and Dr. Rosie Hails (Centre for Ecology and Hydrology, University of Oxford)) aim to address this gap in the theoretical literature by developing mathematical models in continuous time, extending the approach of Ebert & Weisser (1997) to include multiple strains within a host, and to account for competitive dynamics between the strains. These models will be parameterised using data from laboratory experiments. Understanding the within host dynamics of mixed infections is the first step to developing more realistic population models.

Appendix A

Stability

A.1 Linear Stability Analysis

Linear stability analysis of systems of ordinary differential equations of the form

$$\frac{d\mathbf{x}}{dt} = \mathbf{f}(\mathbf{x})$$

which has steady state $\mathbf{x}^*$ given by $\mathbf{f}(\mathbf{x}^*) = 0$, involves linear systems of the form

$$\frac{d\mathbf{x}}{dt} = J\mathbf{x}$$

where J is the Jacobian or community matrix. It can be shown that the steady state $\mathbf{x}^*$ is linearly stable (or just stable) if all the eigenvalues of J have negative real part (see King, Billingham & Otto (2003) for example).
If the system is of n-th order, the characteristic polynomial of J can be written as

$$P(\lambda) = \lambda^n + a_1\lambda^{n-1} + \cdots + a_n = 0 \tag{A.1}$$

where the coefficients a_i, $i = 0, 1, \ldots, n$ are all real.
Thus for (linear) stability we require that all roots of $P(\lambda)$ have $Re(\lambda) < 0$.

A.2 Routh-Hurwitz Criteria for Quadratics

Lemma A.2.1. *If $P(\lambda) = \lambda^2 + a_1\lambda + a_2$, then $Re(\lambda) < 0$ if, and only if, $a_1, a_2 > 0$.*

A.3 Routh-Hurwitz Criteria for Cubics

Lemma A.3.1. *If $P(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$, then $Re(\lambda) < 0$ if, and only if,*

$$a_1 > 0, \quad a_3 > 0, \text{ and } \quad a_1a_2 - a_3 > 0.$$

Bibliography

Anderson, R. M. & May, R. M. (1981). The population dynamics of microparasites and thier invertebrate hosts, *Phil. Trans. R. Soc. Lond. B* **291**: 451–524.

Anderson, R. M. & May, R. M. (1992). *Infectious Diseases of Humans: Dynamics and Control*, Oxford University Press.

Anon (2002). How safe is GM food?, *The Lancet* **360**: 1261.

Antonovics, J. & Thrall, P. H. (1994). The cost of resistance and the maintenance of genetic polymorphism in host-pathogen systems, *Proc. R. Soc. Lond. B* **257**: 105–110.

Bascompte, J. & Solé, R. V. (1994). Spatially induced bifurcations in single-species population dynamics, *J. Anim. Ecol.* **63**: 256–264.

Bascompte, J. & Solé, R. V. (1995). Appropriate formulations for dispersal in spatially structured models: Reply, *J. Anim. Ecol.* **64**: 665–666.

Beddington, J. R. (1975). Mutual interference between parasites or predators and its effects on searching efficiency, *J. Anim. Ecol.* **44**: 331–40.

Begon, M. & Bowers, R. (1995). Beyond host-pathogen dynamics, *in* B. T. Grenfell & A. P. Dobson (eds), *Ecology of Infectious Diseases in Natural Populations*, Cambridge University Press, pp. 478–509.

Boots, M. & Begon, M. (1993). Trade-offs with resistance to a granulosis virus in the indian meal moth examined by a laboratory evolution experiment, *Funct. Ecol.* **7**: 528–534.

Boots, M. & Begon, M. (1995). Strain differences in the indian meal moth, plodia-interpunctella, in response to a granulosis virus, *Res. Popul. Ecol.* **37**: 37–42.

Boots, M. & Haraguchi, Y. (1999). The evolution of costly resistance in host-parasite systems, *Am. Nat.* **153**: 359–370.

Bowers, R. G., Boots, M. & Begon, M. (1994). Life-history trade-offs and the evolution of pathogen resistance: Competition between strains, *Proc. R. Soc. Lond. B* **257**: 247–253.

Bowers, R. G. & White, A. (2002). The adaptive dynamics of lotka-volterra systems with trade-offs, *Math. Biosci.* **175**: 67–81.

Bowers, R. G., White, A., Boots, M., Geritz, S. A. H. & Kisdi, É. (2003). Evolutionary branching/speciation: Contrasting results from systems with explicit or emergent carrying capacities, *Evol. Ecol. Res.* **5**: 883–891.

Britton, N. F. (2003). *Essential Mathematical Biology*, Springer.

Charnley, A. K. (1997). *The Mycota IV: Environmental and Microbial Relationships*, Springer, chapter Entomopathogenic Fungi and Their Role in Pest Control, pp. 185–201. D. T. Wicklow & B. E. Soderstrom, eds.

Chow, S., Mallet-Paret, J. & Shen, W. (1998). Traveling waves in lattice dynamical systems, *J. Differ. Equations* **149**: 248–291.

Comins, H. N., Hassel, M. P. & May, R. M. (1992). The spatial dynamics of host-parasitoid systems, *J. Anim. Ecol.* **61**: 735–748.

Cruywagen, G. C., Kareiva, P., Lewis, M. A. & Murray, J. D. (1996). Competition in a spatially heterogeneous environment: Modelling the risk of spread of a genetically engineered population, *Theor. Pop. Biol.* **49**: 1996.

Dixon, A. F. G. (1973). *Biology of Aphids*, Edward Arnold (Publishers) Limited.

Dixon, A. F. G. (1985). *Aphid Ecology*, Chapman & Hall.

Ebert, D. & Weisser, W. W. (1997). Optimal killing for obligate killers: The evolution of life histories and virulence of semelparous parasites, *Proc. R. Soc. Lond. B* **264**: 985–991.

Ermentrout, G. B. & Edelstein-Keshet, L. (1993). Cellular automata aprroaches to biological modelling, *J. Theor. Biol.* **160**: 97–133.

Evans, H. E. (1984). *Insect Biology*, Addison-Wesey Publishing Company.

Ferrari, J. & Godfray, H. C. J. (2003). Resistance to a fungal pathogen and host plant specialization in the pea aphid, *Ecol. Lett.* **6**: 111–118.

Ferrari, J., Muller, C. B., Kraaijeveld, A. R. & Godfray, H. C. J. (2001). Clonal variation and covariation in aphid resistance to parasitoids and a pathogen, *Evolution* **55(9)**: 1805–1814.

Funasaki, E. & Kot, M. (1993). Invasion and chaos in a periodically pulsed mass-action chemostat, *Theor. Popul. Biol.* **44**: 203–224.

Geritz, S. A. H., Kisdi, É., Meszéna, G. & Metz, J. A. J. (1998). Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree, *Evol. Ecol.* **12**: 35–57.

Gillespie, J. H. (1975). Natural selection for resistance to epidemics, *Ecology* **56**: 493–495.

Gurney, W. S. C. & Nisbet, R. M. (1998). *Ecological Dynamics*, Oxford University Press.

Hanski, I. & Simberloff, D. (1997). The metapopulation approach, its history, conceptual domain, and its application to conservation biology, *in* I. Hanski & M. E. Gilpin (eds), *Metapopulation Biology*, London: Academic Press.

Hardin, G. (1968). The tragedy of the commons, *Science* **162**: 1243–1248.

Harris, K. F. & Maramorosch, K. (eds) (1982). *Pathogens, Vectors and Plant Diseases*, Academic Press.

Hart, D. R. & Gardner, R. H. (1997). A spatial model for the spread of invading organisms subject to competition, *J. Math. Biol.* **35**: 935–948.

Hassell, M. P. (1974). Density dependence in single species populations, *J. Anim. Ecol.* **44**: 283–296.

Hassell, M. P. (1980). Foraging strategies, population models and biological control: A case study, *J. Anim. Ecol.* **49**: 603–28.

Hassell, M. P. (2000). Host-parasitoid population dynamics, *J. Anim. Ecol.* **69**: 543–566.

Hassell, M. P., Miramontes, O., Rohani, P. & May, R. M. (1995). Appropriate formulations for dispersal in spatially structured models: Comments on bascompte & solé, *J. Anim. Ecol.* **64**: 662–664.

Hill, D. S. (1997). *The Economic Importance of Insects*, Chapman & Hall.

Hochberg, M. E. (1991). Non-linear transmission rates and the dynamics of infectious disease, *J. Theor. Biol.* **153**: 301–321.

Hodson, D. J., Hitchman, R. B., Vanbergen, A. J., Hails, R. S., Hartley, S. E., Possee, R. D., Watt, A. D. & Cory, J. S. (2003). The existence and persistence of genotypic variation in nucleopolyhedrovirus populations, *in* R. S. Hails, J. Beringer &

H. C. J. Godfray (eds), *Genes in the Environment*, Vol. BES Symposium Volume 15, Blackwells, pp. 258–280.

Holland, J. M., Perry, J. N. & Winder, L. (1999). The within-field spatial and temporal distribution of arthropods in winter wheat, *B. Entomol. Res.* **89**: 499–513.

Holland, J. M., Winder, L. & Perry, J. N. (2000). The impact of dimethoate on the spatial distribution of beneficial arthropods in winter wheat, *Ann. Appl. Biol.* **136**: 93–105.

Holt, R. D. & Pickering, J. (1985). Infectious disease and species coexistence: A model of lotka-volterra form, *Am. Nat.* **126**: 196–211.

Hoy, M. A. (1998). Myths, models and mitigation of resistance to pesticides, *Phil. Trans. R. Soc. Lond. B* **353**: 1787–1795.

Iooss, G. & Joseph, D. D. (1980). *Elementary Stability and Bifurcation Theory*, New York: Springer-Verlag.

Jansen, V. A. A. & Lloyd, A. L. (2000). Local stability analysis of spatially homogeneous solutions of multi-patch systems, *J. Math. Biol.* **41**: 232–252.

Kaneko, K. (1992). Overview of coupled map lattices, *Chaos* **2**: 279–282.

Kermack, W. O. & McKendrick, A. G. (1927). Contributions to the mathematical theory of epidemics, *Proc. R. Soc. London A* **115**: 700–721.

King, A. C., Billingham, J. & Otto, S. R. (2003). *Differential Equations*, Cambridge University Press.

Kisdi, É. & Meszéna, G. (1995). Life history with lottery competition in a stochastic environment: ESSs which do not prevail, *Theor. Pop. Biol.* **47**: 191–211.

Kot, M. (1992). Discrete-time traveling waves: Ecological examples, *J. Math. Biol.* **30**: 413–436.

Lacey, L. A., Frutos, R., Kaya, H. K. & Vail, P. (2001). Insect pathogens as biological control agents: Do they have a future?, *Biol. Control* .

Langa, J. A., Robinson, J. C. & Suárez, A. (2003). Forwards and pullback behaviour of a non-autonomous lotka-volterra system, *Nonlinearity* **16**: 1277–1293.

Levins, R. (1969). Some demographic and genetic consequences of environmental heterogeneity for biological control, *Bull. Entomol. Soc. Am.* **15**: 237–240.

Levins, R. (1970). Extinction, *in* M. Gerstenhaber (ed.), *Some Mathematical Problems in Biology*, Providence, RI American Mathematical Society, pp. 77–107.

Liu, W., Hethcote, H. W. & Levin, S. A. (1987). Dynamical behavior of epidemiological models with nonlinear incidence rates, *J. Math. Biol.* **25**(4): 359–380.

May, R. M. (1978). Host-parasitoid systems in patchy environments: A phenomenological model, *J. Anim. Ecol.* **47**: 833–43.

May, R. M. & Oster, G. F. (1976). Bifurcations and dynamic complexity in simple ecological models, *Am. Nat.* **110**: 573–599.

Maynard Smith, J. & Price, G. R. (1973). The logic of animal conflict, *Nature* **246**: 15–18.

Metz, J. A. J., Nisbet, R. & Geritz, S. A. H. (1992). How should we define 'fitness' for genernal ecological scenarios?, *Trends Ecol. Evol.* **7**: 198–202.

Milks, M. L. & Theilmann, D. A. (2000). Serial selection for resistance to a wild-type and to a genetically modified nucleopolyhedrovirus in *trichoplusia*, *Biol. Control* **19**: 283–289.

Milner, R. J. (1982). On the occurrence of pea aphids, *acyrthosiphon pisum*, resistant to isolates of the fungal pathogen *erynia neoaphidis*, *Entomol. Exp. Appl.* **32**: 23–27.

Milner, R. J. (1985). Distribution in time and space of resistance to the pathogenic fungus *erynia neoaphidis* in the pea aphid *acyrthosiphon pisum*, *Entomol. Exp. Appl.* **37**: 235–240.

Moran, P. A. P. (1950). Some remarks on animal population dynamics, *Biometrics* **6**: 250–258.

Murray, J. D. (1989). *Mathematical Biology*, Springer.

Neubert, M. G., Caswell, H. & Murray, J. D. (2002). Transient dynamics and pattern formation: Reactivity is necessary for turing instabilities, *Math. Biosci.* **175**: 1–11.

Neubert, M. G., Kot, M. & Lewis, M. A. (1995). Dispersal and pattern formation in a discrete-time predator-prey model, *Theor. Popul. Biol.* **48**: 7–43.

Nicholson, A. J. & Bailey, V. A. (1935). The balance of animal populations, *Proc. Zool. Soc. London* **3**: 551–598.

Paoletti, M. G. & Pimentel, D. (2000). Environmental risks of pesticides versus genetic engineering for agricultural pest control, *J. Agr. Environ. Ethic.* **12**: 279–303.

Pollan, M. (1998). Playing god in the garden, New York Times. Appeared in The New York Times on Sunday, October 25, 1998.

Ricker, W. E. (1954). Stock and recruitment, *J. Fish. Res. Board Can.* **11**: 559–623.

Roditakis, E. (1999). *Interactions Between the Peach Aphid Myzus persicae and the Entomopathogenic Fungus Verticillium lecanii*, PhD thesis, University of Bath.

Rogers, D. J. (1972). Random search and insect population models, *J. Anim. Ecol.* **41**: 369–83.

Rohani, P., May, R. M. & Hassell, M. P. (1996). Metapopulations and equilibrium stability: The effects of spatial structure, *J. Theor. Biol.* **181**: 97–109.

Rohani, P. & Ruxton, G. D. (1999a). Dispersal and stability in metapopulations, *IMA J. Math. Appl. Med.* **16**: 297–306.

Rohani, P. & Ruxton, G. D. (1999b). Dispersal-induced instabilities in host-parasitoid metapopulations, *Theor. Popul. Biol.* **55**: 23–36.

Sabin, G. C. W. & Summers, D. (1992). Chaos in a periodically forced predator-prey ecosystem model, *Math. Biosci.* **113**: 91–113.

Scott, J. G. (1990). *Pesticide Resistance in Arthropods*, Chapman & Hall, chapter Investigating Mechanisms of Insecticide Resistance: Methods, Strategies, and Pitfalls. R. T. Roush & B. E. Tabashnik, eds.

Segel, L. A. & Jackson, J. L. (1972). Dissipative structure: An explanation and an ecological example, *J. Theor. Biol.* **37**: 545–559.

Sherratt, J. A. (1995). Diffusion-driven instabilities in oscillating environments, *Eur. J. Appl. Math.* **6**: 373–381.

Shulgin, B., Stone, L. & Agur, Z. (1998). Pulse vaccination strategy in the SIR epidemic model, *B. Math. Biol.* **60**: 1–26.

Stearns, S. C. (1992). *The Evolution of Life-Histories*, Oxford University Press.

Tanada, Y. & Kaya, H. K. (1993). *Insect Pathology*, Academic Press.

Tilman, D. & Kareiva, P. (eds) (1997). *Spatial Ecology*, Princeton University Press.

Turing, A. M. (1952). The chemical basis of morphogenesis, *Phil. Trans. R. Soc. Lond. B* **237**: 37–72.

Wade, F. A. & Leather, S. R. (2002). Overwintering of the sycamore aphid, *drepanosiphum platanoidis*, *Entomol. Exp. Appl.* **104**: 241–253.

Wang, M. H., Kot, M. & Neubert, M. G. (2002). Integrodifference equations, allee effects, and invasions, *J. Math. Biol.* **44**: 150–168.

White, K. A. J. & Wilson, K. (1999). Modelling density-dependent resistance in insect-pathogen interactions, *Theor. Popul. Biol.* **56**: 163–181.

Winder, L., Alexander, C. J., Holland, J. M., Woolley, C. & Perry, J. N. (2001). Modelling the dynamic spatio-temporal response of predators to transient prey patches in the field, *Ecol. Lett.* **4**: 568–576.